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DOES DISSEMINATED SCLEROSIS OCCUR IN THE BANTU?

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M. Gelfand[1] states that disseminated sclerosis does not occur in the Bantu. F. M. K. Muwazi and H. C. Trowell[2] in their comprehensive review of Neurological Disease among African Natives of Uganda, state 'we have never encountered any of the demyelinating diseases of the nervous system. It is true a few cases of disseminated sclerosis have been reported in Africans in West Africa, yet the case records and investigations were somewhat restricted and the autopsy material scanty'.

B. Goldstein[3] reported 2 cases in East African Natives, one disseminated in type, the other of the progressive spastic paraplegic type seen in middle-aged persons.

Craig Cochran[4] reported a case in a non-European female of Xosa and Coloured extraction.

CASE RECORD

The following is the case record of a patient, of pure Bantu descent (daughter of a Zulu father and a Msutu mother) who presents the clinical picture of disseminated schlerosis.

M.M., aged 26, employed in a bakery, was first admitted on 26 July 1955 with the following complaints:

For a month before admission she had been walking 'like a baby'. She had noticed that on looking to the right she saw double and on turning her head to the right she experienced dizziness. This dizziness would occasionally cause her to fall to the ground. There had been a tendency over the previous month to drop objects, more so from the left hand.

Before this the patient had been quite well, except that over the past year she had occasional headaches, which if anything had tended to diminish in severity and frequency.

The findings on examination were: A fine nystagmus to the right and a coarser one to the left; diminished corneal reflex on the left. The left limbs were hypotonic, with a marked intention tremor and incoordination of alternating movements in the left hand. The patient showed similar cerebellar signs on the right, but much milder in degree.

The remainder of a detailed neurological examination was entirely negative.

The following investigations were carried out soon after admission:

X-rays of chest and skull were negative.

Full blood-count showed no abnormality.

Lumbar puncture; pressure 150 mm. of water and a satisfactory response to jugular-vein compression; cells—2 lymphocytes per c.mm.; chlorides 690 mg.%, sugar 44 mg.%, protein 118 mg.%; Wassermann reaction negative.

Wassermann reaction of the blood was negative.

During the next few weeks while in hospital the patient complained of precipitancy.

On 19 August a vertebral angiogram showed no displacement of the vessels and no abnormal filling defects. Shortly afterwards her condition began to improve and by the end of August she no longer complained of diplopia or dizziness and the cerebellar signs, though still present, were greatly diminished.

In October the cerebellar signs again became marked and in addition the patient developed totally new symptoms and signs. She complained of excessive salivation and dysphagia and her speech was markedly slurred. The excessive salivation and dysphagia subsided after a week, but the slurring of speech became worse. There was weakness of both lower limbs, so much so that she could not support herself. Both the knee and ankle jerks were greatly increased, ankle clonus being elicited on the left side; the spasticity was more marked on the left side. Gradually the spastic paraparesis regressed, but at the end of November the patient developed a marked weakness and spasticity of the whole of the left side, more marked in the face than the arm, and in the arm than the leg. By the end of December the spastic hemiparesis of the left side was the dominant lesion.

On 19 November an air encephalogram was carried out and showed that the fourth, third and lateral ventricles and the aqueduct of Sylvius were of normal size, shape and position. In none of the pictures were there any abnormal filling defects. On

26 November audiogram and caloric tests were carried out and found to be normal.

The patient again improved and towards the end of January 1956 she was discharged. At this stage her signs and symptoms were decreasing and she was just barely ambulant with help. Except for a few days in December during which the right pupil was found to be smaller than the left (but both pupils reacted to light and accommodation) she had developed no further signs.

The patient was readmitted for follow-up on 5 April 1956 when the findings were as follows: Slurring of speech, nystagmus to the right as well as to the left—coarse in both directions. A fine vertical nystagmus on upward gaze. The corneal reflexes were now brisk and equal on both sides. There was bilateral spasticity and cerebellar signs, both more marked on the left than on the right.

Now, however, in addition to these signs there was a loss of joint position sense in the right big toe. This sensory abnormality disappeared completely towards the end of April and about this time the inequality of the pupils, as previously described, reappeared for a few days.

Throughout the patient's stay in hospital her mental state was one of euphoria.

At the time of her discharge in the middle of May 1956 she was able to walk the length of the ward by means of holding on to beds or using crutches.

Repeated cerebrospinal-fluid studies showed negative Wassermann reactions, protein which ranged from 110 to 150 mg.%, and a negative colloidal-gold curve.

DIFFERENTIAL DIAGNOSIS

'Moderately severe mixed case is likely to be confounded only with diseases whose morbid processes are diffused; and of these cerebro-spinal syphilis alone need be considered. Clinical resemblance between the two is often intimate, but a history of the latter may be forthcoming, and in any case laboratory tests suffice to distinguish'.[5]

Against a diagnosis of acute disseminated encephalomyelitis are the absence of a history of vaccination or any infectious fever, the bright intelligence as against the post-encephalic 'vegetable', and the dissemination in time and place. In the non-European bizarre nervous presentations of avitaminosis are always to be considered.[6] This patient was extremely well nourished on admission and the dissemination in time and place occurred while she was in hospital on a well balanced diet.

Tumour infiltrating the base of the brain may simulate disseminated sclerosis.[7] The normal CSF pressure with satisfactory response to jugular-vein compression, the absence of increased intracranial pressure, and the normal findings of the special investigations, are against this diagnosis; as is the course of the disease as described in our patient.

Two points differ from the text-book description of disseminated sclerosis, viz. (1) the negative colloidal-gold curve, and (2) the protein in the CSF ranging from 110-150 mg.%. Nevertheless, these differences are compatible with the diagnosis of disseminated sclerosis.

Differences in incidence of positive colloidal-gold curves result from minor differences in technique. In the City Hospital of New York a 60-70% incidence of a positive colloidal-gold curve was obtained in disseminated sclerosis;[8] whereas at the Neurological institute of New York the incidence was 10-20%. It is generally agreed that the percentage of positive curves in routine clinical laboratories in disseminated sclerosis is low.[8]

McAlpine[9] in 512 cases found a protein greater than 100 in 5 cases. A protein as high as 255 has been recorded in disseminated sclerosis.[10] A high protein is found in 32% of cases in the initial stages of the disease.[10] Picht, Seuberling, Gaupt and Schroeder found the percentage of CSF abnormalities to be directly proportional to the severity of the clinical picture.[10] The high CSF protein in our case may well be associated with the rapidly progressive severity of the condition.

SUMMARY

A case is described in a young Bantu woman with a clinical picture typical of disseminated sclerosis. If the patient was a European no other diagnosis would have been entertained. In the Bantu, however, one accepts this diagnosis with reservation and awaits further developments.

SAMEVATTING

'n Klinies tipiese geval van verspreide sklerose in 'n jong Bantu vrou word beskryf. As dit 'n Blanke pasiënt was, sou hierdie diagnose algemeen aangeneem word. In die Bantu egter is die diagnose minder seker en moet 'n mens wag op verdere verwikkelings.

We wish to acknowledge thanks to Drs. S. M. Stone and G. W. Brammer of Germiston, who referred the case to us and kept us in contact with the patient. We wish to thank Dr. G. D. Elliot, Acting Superintendent of Baragwanath Hospital, for permission to publish this case.

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UNION DEPARTMENT OF HEALTH BULLETIN

Union Department of Health Bulletin. Report for the 7 days ended 16 August 1956.

Plague, Smallpox, Typhus Fever: Nil.

Epidemic Diseases in Other Countries.

Plague: Nil.

Cholera in Chalna (Pakistan).

Smallpox in Rangoon (Burma); Bombay, Karikal, Madras, Pondicherry, Visakhapatnam (India); Makassar (Indonesia); Dacca, Lahore (Pakistan); Mombasa (Kenya).

Typhus Fever in Alexandria, Cairo (Egypt).

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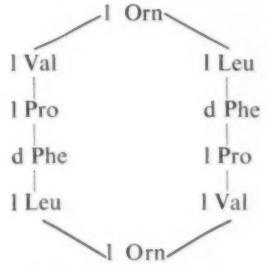
VAN DIE REDAKSIE

PEPTIED-ANTIBIOTIKA

Parallel met ondersoek na die struktuur van die polipeptiedprinsipes van die harsingslymklier,[1] vind navorsing op 'n soortgelyke grondslag plaas om die struktuur van die polipeptied-antibiotika[2] vas te stel.

Tirotrisien, die eerste bakteriese antibiotika wat verkry was (van *Bacillus brevis*), is naderhand in gramisidiene en tirosidiene geskei. Daar is getoon dat een hiervan, gramisidien-S, uit 10 aminosuur-eenhede bestaan, en die wyse waarop hulle aaneenskakel, is bekend. Daar is twee molekule elk van l-ornitien, l-valien, l-leusien, l-prolien en d-fenielalanien. Die volledige struktuur van hierdie antibiotika word as volg beskrywe.



Die struktuur van tirosidien-A en tirosidien-B is ook bekend en toon 'n groot ooreenkoms met dié van gramisidien-S. Hulle is almal ringvormige dekapeptiede, met 5 van die aminosure, in elk, identies en in dieselfde volgorde aaneengeskakel. Elkeen bevat d-fenielalanien en nie die l-isomeer nie wat gewoonlik in proteïene aanwesig is. Dit is aan die hand gedoen dat hierdie eienskap miskien die antibakteriese aktiwiteit van hierdie besondere polipeptied-antibiotika verklaar. Die ringvormige aard van die polipeptiede is nie noodsaaklik vir antibakteriese aktiwiteit nie, aangesien 'n reguitketting-dekapeptied, met dieselfde aminosuur-volgorde, gesintetiseerd is, wat 1/10de tot 1/40ste van die antibakteriese aktiwiteit van gramisidien-S[3] besit. Tirosidiene en gramisidien-S is ietwat meer ingewikkeld in struktuur as die oktapeptiede vasopressien en oksitosien.

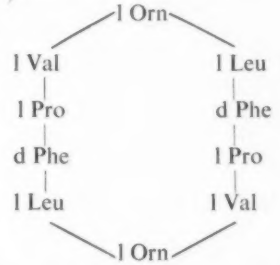
Dit kom voor asof die verskillende antibiotika wat deur *Bacillus subtilis* geproduseer word, ook polipeptiede is. Dit is bewys dat basitrasien byvoorbeeld, uit 10 verskillende polipeptiede bestaan, waarvan 3 antibakteries is. Die volledige struktuur van basitrasien-A is nog nie vasgestel nie maar die aminosuur-volgorde van 'n gedeelte van die molekuul is bekend.

EDITORIAL

PEPTIDE ANTIBIOTICS

Parallel with the investigations into the structure of the polypeptide principles of the pituitary gland,[1] work has been proceeding along similar lines to determine the constitution of the polypeptide antibiotics.[2]

Tyrothricin, the first bacterial antibiotic to be obtained (from *Bacillus brevis*) was separated subsequently into gramicidins and tyrocidines. One of these, gramicidin S, has been shown to consist of 10 amino-acid units, and the way in which they are linked together is known. There are 2 molecules each of l-ornithine, l-valine, l-leucine, l-proline and d-phenylalanine. The complete structure of this antibiotic is depicted as follows:



The structure of tyrocidine A and tyrocidine B is also known, and closely resembles that of gramicidin S. They are all cyclic decapeptides, with 5 of the amino acids in each identical and linked together in the same sequence. Each contains d-phenylalanine, not the l isomer, which is commonly present in proteins. It has been suggested that this feature may account for the antibacterial activity of these particular polypeptide antibiotics. The cyclic nature of the polypeptides is not essential for antibacterial activity, since a straight-chain decapeptide with the same amino-acid sequence has been synthesized and found to have 1/10th to 1/40th the antibacterial activity of gramicidin S.[3] The tyrocidines and gramicidin S are somewhat more complicated in structure than the octapeptides vasopressin and oxytocin.

The several antibiotics produced by *Bacillus subtilis* appear also to be polypeptides. Bacitracin, for example, has been shown to consist of 10 different polypeptides, of which 3 have antibacterial potency. The complete structure of bacitracin A is not yet established, but the amino-acid sequence of part of the molecule is known.

The group of peptide antibiotics obtained from

Die peptied-antibiotika-groep wat van *Bacillus subtilis* verkry word, staan as polimiksiene bekend. Hulle struktuur is gedeeltelik gestaaf. Dit is getoon dat polimiksien 5 bestanddele het waarvan die vernaamste een, polimiksien-A, identies met erosporien is.

Die samestelling van ander peptiede met antibiotiese aktiwiteit word ondersoek, terwyl die sintese van sulke middele soos die reguitketting-dekapeptied, waarna hierbo verwys is, van groot belang is.

Slegs gedurende die afgelope paar jare is die pogings, om die struktuur van die polipeptiede toe te lig, geslaagd. Die metode van papierchromatografie eerder as die ouer tegnieke, het gehelp om die konstituerende aminosure makliker te skei. Verder, deur gebruik te maak van verwante metodes van navorsing, kan die volgorde van die aminosure in die ketting vasgestel word, asook hulle aard en die relatiewe getalle van elk wat in die verbinding aangetref word. Met sommige van die metodes wat in hierdie studies gebruik is, word die end-aminosuur uitgeken; sekere van hierdie metodes stel navorsers in staat om die polipeptied trapsgewyse af te breek. Deur middel van papierchromatografie is die produkte van parsiële hidrolise van volledige polipeptied-antibiotika al van mekaar geskei; die kolletjies op die papier word afsonderlik ge-eludeer, dan word die di- en tripeptiede heeltemal gehidroliseer en die aminosure word deur verdere papierchromatografie[2] geïdentifiseer. Waar die polipeptied by ondersoek nie vry is van peptied-onsuiwerhede nie, moet ander metodes gebruik word. Een van die metodes wat vandag algemeen gebruik word om die polipeptiede van mekaar te skei, is dié van teenstroomverdeling: die mengsel word agtereenvolgens in 'n spesiale apparaat verdeel wat 'n oplossings-vloeistof-fase en 'n waterfase bevat; daar is 'n aantal buise so gerangskik dat komponente met 'n hoë oplos-middel/water-verdelingskoëffisiënt by die een en tvan die apparaat bymekaar kom en dié met 'n lae verdelings-koëffisiënt by die ander ent. Tirotrisien was deur hierdie metode in verskeie verwante polipeptiede gefraksioneer. Papierelektroforese is nog 'n metode wat gebruik word om verwante polipeptiede van mekaar te skei.

Deur gebruik te maak van die nuwer en meer aantreklike tegnieke wat dit moontlik maak om stowwe te skei en te ontleed sonder om hulle unieke en labiele strukture te versteur, het navorsing i.v.m. polipeptiede die afgelope jare vinnig gevorder.

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Bacillus polymyxa are known as polymyxins. Their structure is partly established. Polymyxin has been shown to have 5 constituents, of which the main one, polymyxin A, is identical with aerosporin.

The structure of other peptides having antibiotic activity is being investigated, while the synthesis of such agents as the straight-chain decapeptide to which reference is made above, is of great interest.

It is only in the last few years that attempts to elucidate the structure of polypeptides have been successful. The method of paper chromatography has enabled the constituent amino-acids to be separated more easily than by the older techniques. Furthermore, by the use of related methods of investigation the sequence of the amino acids in the chain can be determined, as well as their nature and the relative numbers of each present in the compound. Among the methods that have been used in these studies are some in which the terminal amino-acid is identified; certain of the methods permit stepwise degradation of the polypeptide to be effected. By paper chromatography the products of partial hydrolysis of complete polypeptide antibiotics have been separated from one another; the spots on the paper are separately eluted, the di- and tri- peptides then hydrolysed completely, and the amino acids identified by further paper chromatography.[2] Where the polypeptide under investigation is not free from peptide impurities other methods must be employed. One that is widely used at present, to separate polypeptides from one another, is the method of counter-current distribution, in which the mixture is successively partitioned in a special apparatus containing a solvent phase and an aqueous phase; there are a number of tubes so arranged that components with a higher solvent water partition-coefficient collect at one end of the apparatus and those with a low partition-coefficient accumulate at the other end. By this method tyrothricin was fractionated into several related polypeptides. Paper electrophoresis is another method used to separate related polypeptides from one another.

The investigation of polypeptides has advanced rapidly in recent years through the application of the newer elegant techniques which make it possible to separate and analyse substances without disturbing their unique and labile structures.

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THE EARLY DIAGNOSIS OF ACUTE ABDOMINAL EMERGENCIES IN INFANCY AND EARLY CHILDHOOD

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It is not generally appreciated that acute abdominal emergencies are common in infancy and early childhood. Although chemotherapy and antibiotics, improved techniques in pre- and post-operative care, and better anaesthesia have been reponsible for a steady and reassuring decline in the annual mortality rate from these emergencies, there is still much room for improvement. The reduction in mortality has not equalled that found in adults, and disconcerting facts are the alarming death rate from neonatal obstructions still prevalent in many centres, the frequency of irreducible intussusception, and the persistently high fatality-rate from acute appendicitis in children under the age of 5 years.

It is the purpose of this paper to ascertain the causes of the higher mortality figures in small children, and to consider the distinctive clinical features of the commoner conditions which present as acute abdominal emergencies in childhood. The data, which are based mainly on personal experience, are correlated with the views expressed in current literature and illustrated by an analysis of 314 abdominal emergencies treated in our Children's Surgical Department during the 2-year period January 1953 to December 1954.

The Causes of the Mortality

There are two main factors responsible for the higher mortality-rate in children, viz. the rapid progress of disease processes in the very young and delay in diagnosis. This is best illustrated by reference to 3 common surgical emergencies, viz. neonatal obstructions, acute intussusception and acute appendicitis.

Neonatal Obstructions. Even in simple obstructions necrotic changes may occur in the bowel wall within 24 hours and the mortality rises steeply with every day's delay in treatment until a figure of 80% is reached on the 5th day. This must be coupled with the fact that over 30% of our cases were admitted on the 5th day and later.

Acute Intussusception. As a rule the viability of the bowel is seriously affected when the intussusception has been present for 48 hours and the mortality rises from practically *nil* with treatment in the first 24 hours to approximately 40% if treatment is delayed until the 4th day. The *average* duration of symptoms in our cases was 46 hours.

Acute Appendicitis. The percentage of cases with rupture of the appendix rises with every hour that passes. Approximately 10% may perforate in the first 12 hours, and this figure rises to 30% after 24 hours and 70% after 48 hours. The mortality of ruptured appendicitis is approximately 5 times that of uncomplicated appendicitis. In 52% of our cases the organ had already ruptured, with either a localized abscess or diffuse peritonitis on admission.

Delay in Diagnosis

From the above it should be obvious that delay in diagnosis is the most important preventable factor concerned, and before we consider the symptomatology of the various surgical emergencies it would be as well to analyse the reasons for delay. These may be classified under the following headings:

1. *Delay in Calling in the Advice of a Doctor.* This is one of the most important reasons. Vomiting is so common in newborn infants that midwives are seldom concerned about it, and in this they are often aided and abetted by irresponsible teachers who fail to stress the possible grave significance of persistent vomiting in the newborn. In older children a minor 'upset tummy' is equally common and parents get used to the idea that teething powders, castor oil, vermifuges, etc., will cure it. The result is that the ominous belly-ache or episode of vomiting which heralds the onset of a serious surgical emergency is often neglected until the child is desperately ill.

2. *Inaccuracy of the History.* An accurate description of the symptoms by the child is often sorely lacking owing to his very limited powers of description. At the same time the parent is often equally unreliable, tending to be either over-anxious or too matter-of-fact.

3. *Difficulties in Eliciting and Interpreting Physical Signs.* 'The common practice of starting the examination with the child's head, gagging him with a throat stick, upsetting him with a cold stethoscope and then pummelling his belly with a heavy hand, always fails to elicit the important physical signs.' (Gross, 1953). Furthermore, it is often forgotten that children react differently from adults to disease processes. This applies particularly to the child's reaction to pain. Some may wince, others whimper and others cry and shout, and yet others may lie quietly in bed. Also, vomiting occurs more readily than in adults and is often the most prominent symptom, while diarrhoea occurs in conditions which, in adults, are associated with constipation. High fever occurs even apart from acute infections, localized tenderness and rigidity may be absent, and rectal examination is often misleading in regard to pelvic tenderness.

4. *A Lack of Awareness on the Part of the Medical Profession* that surgical emergencies are indeed common in small children. In recent years the indiscriminate prescribing of antibiotics has led to even more errors, because these drugs tend to mask the true nature of the underlying disease until complications have arisen.

THE SURGICAL EMERGENCIES

Fig. 1 reflects under 11 headings the various abdominal

surgical conditions that were treated over the period of 2 years and the respective mortality (dark portions of graphs). Table I gives some of the details of the rarities

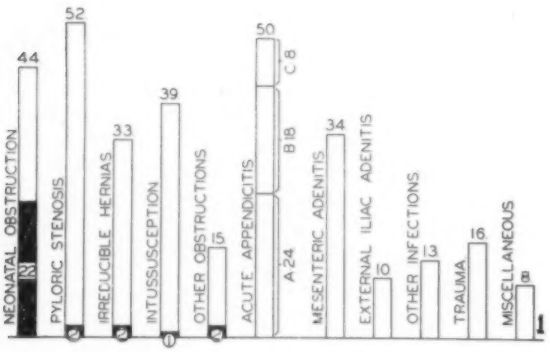

Fig. 1. Acute abdominal emergencies—children of 10 years and under. 314 cases.

TABLE I

Other Obstructions		
Volvulus	4	(2 due to Meckel's diverticulum).
Lymphoma	3	
Mesenteric cyst	2	
Tuberculosis	2	
Bands and adhesions	3	(1 due to Meckel's diverticulum).
Other Infections		
Tuberculous mesenteric adenitis	4	
Meckel's diverticulitis	2	
Primary peritonitis	2	
Hepatic abscess	2	
Perinephric abscess	1	
Crohn's disease	1	
Foreign body	1	
Abdominal Trauma		
Penetrating wounds	5	
Ruptured bowel	2	
Ruptured spleen	2	
Ruptured kidney	3	
Closed injury—no visceral damage	2	
Haematoma of abdominal wall	2	
Miscellaneous		
Haemorrhage from Meckel's diverticulum	2	
Haemorrhage from liver infested with flukes	1	
Torsion of ovarian cyst	2	
Round-worm obstruction of common duct	1	
Acute pancreatitis	1	
Biliary peritonitis	1	

(under 4 of these headings) which will not be discussed. It is of interest that 7 of these cases suffered from complications of a Meckel's diverticulum which should always be kept in mind when a child presents with severe intestinal haemorrhage or persistent abdominal pain.

Acute non-specific mesenteric adenitis and acute suppurative external iliac adenitis have been included (as 2 headings) in Fig. 1 because these conditions are so frequently confused with surgical emergencies especially with acute appendicitis. 50% of our cases of mesenteric adenitis were submitted to laparotomy because of doubt in the diagnosis, and 70% of the cases of iliac adenitis eventually required surgical drainage.

The remaining 5 conditions, viz., neonatal obstructions, pyloric stenosis, irreducible hernia, acute intussusception and acute appendicitis accounted for 2/3rds of our surgical emergencies and will be considered in some detail. The first 3 will be discussed only briefly, while the 2 most important conditions in practice, viz., intussusception and appendicitis will be dealt with at some length.

NEONATAL INTESTINAL OBSTRUCTION

One newborn baby in every 1,000 develops acute intestinal obstruction at, or soon after, birth and will present symptoms within the first few days of life. There are numerous causes of such obstruction, the commonest being ano-rectal malformations and malrotation of the midgut, with or without volvulus and atresia. These infants will die unless treated surgically, and if operation is delayed beyond 48 hours the mortality becomes appalling (Table II). Early diagnosis by the practitioner is thus of paramount importance, and in this connection the following observations will be of value:

TABLE II. NEONATAL OBSTRUCTION: AGE AT OPERATION IN 39 URGENT CASES

Age in hours	*No. of cases*	*Deaths*	*Mortality*
Less than 24	5	1	20%
24– 48	7	3	43%
48– 72	8	4	50%
72– 96	5	3	60%
96–120	5	3	60%
120–144	3	2	67%
Over 144	6	5	83%

1. Repeated or bile-stained vomiting in a newborn infant is always serious and demands prompt hospitilization to exclude intestinal obstruction.

2. Routine examination of the anus and rectum for malformations at birth will reveal the cause in approximately 1/3rd of the cases suffering from an obstructive lesion.

3. The passage of meconium does not exclude a diagnosis of obstruction; it may occur even with complete occlusions.

4. Abdominal distension is often not present in the early stages of high obstructions, and in duodenal occlusions may not appear for several days.

5. Visible peristalsis is a valuable physical sign, but is present only in a minority of cases.

6. The baby's general condition may remain apparently good for several days even in complete obstructions.

Many of the infants can be cured by surgical operation and in some centres the over-all mortality has already dropped to 20%, but the most essential factor that makes for success is early diagnosis.

PYLORIC STENOSIS

Whereas intestinal obstruction presents with symptoms in the first few *days* of life, pyloric stenosis rarely causes symptoms until the infant is some *weeks* old (Fig. 2). In other words, if an infant presents with vomitting in

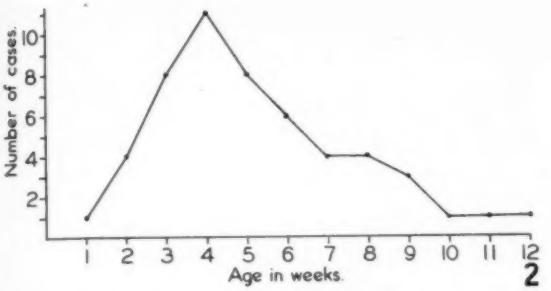

Fig. 2. Pyloric stenosis—age incidence. 52 cases.

the first week of life always regard intestinal obstruction as a more likely possibility than pyloric stenosis. From the second week onwards pyloric stenosis is the more likely possibility, although partial obstructions must also be considered.

Although the diagnosis of pyloric stenosis, which is based on the triad, projectile vomiting, visible peristalsis and a palpable tumour, is usually not difficult, cases are not infrequently neglected simply because a tumour cannot be felt. Such neglected cases become serious emergencies and it is among them that all the deaths occur. The tumour is often difficult to feel and a great deal of patience and experience are required. Occasionally it is more easily detected from the left. Another useful hint is to feed the baby until he vomits, because this is followed by a momentary period of relaxation of the abdominal muscles. Visible peristalsis is also best elicited during a feed. However, it is important not to delay treatment and if an infant *behaves* like one suffering from pyloric stenosis, he should be hospitalized regardless of the presence or absence of physical signs. The practice of trying the effect of Eumydrin must be condemned.

The mortality of pyloric stenosis should not exceed 1%; in experienced hands Ramstedt's operation can offer even better results, provided the diagnosis is not

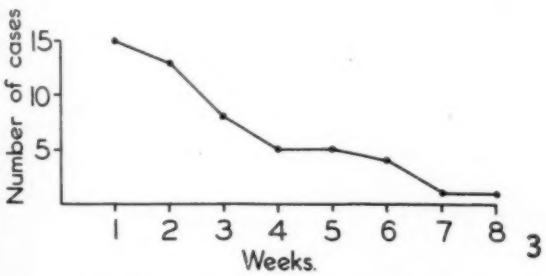

Fig. 3. Pyloric stenosis—duration of symptoms. 52 cases.

delayed. In our cases the duration of symptoms before admission to hospital leaves a good deal of room for improvement (Fig. 3).

STRANGULATED HERNIA

Approximately 6% of infants with inguinal hernia require emergency herniotomy for irreducibility. During the 2 years we had 33 cases who required emergency operations while another 28 irreducible hernias responded to conservative treatment. There were no cases of irreducible umbilical hernia; 90% were boys.

Congenital hernias tend to become irreducible in the first 2 years of life, with the peak incidence during the first 6 months (Fig. 4)—our youngest case was 5 days

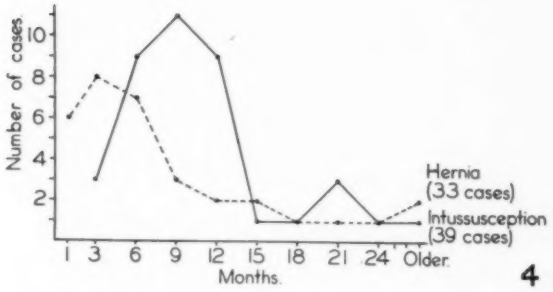

Fig. 4. Irreducible hernia and intussusception—age incidence. 72 cases.

old. Simple irreducibility is much commoner than actual strangulation but the latter is particularly liable to occur in small hernias of recent appearance during the first 6 months of life and may lead to gangrene in less than 12 hours. The bowel was gangrenous in 4 of our cases and they were all under 6 months of age. Two of these died—both were premature infants of less than a month old.

The diagnosis is not always obvious, because the parents may be unaware of the hernia and not infrequently abdominal pain and vomiting are the only symptoms. Mistakes will be avoided only if we adhere to the golden rule that all hernial sites must be examined in any patient presenting with an 'acute abdomen'. Inspection alone is not sufficient—a very small hernia may not be seen, especially in a fat baby, but the tense, tender swelling of an irreducible hernia can always be felt. Local redness and oedema as well as obstructive vomiting and abdominal distension are indications that strangulation has occurred.

ACUTE INTUSSUSCEPTION

Like irreducible hernia, acute intussusception tends to affect babies in the first 2 years of life, particularly between the ages of 3 months and 18 months, with a peak incidence at 9 months (Fig. 4). Although it is rare in the first 3 months it may occur in neonates and has been described in an infant aged 70 hours. Acute intussusception is a particularly dangerous emergency and demands diagnosis within the first 24 hours if the mortality is to be kept down. Yet in 20% of our patients the intussusception was already irreducible and the average duration of symptoms was 46 hours.

It has been stated that the classical text-book description of intussusception portrays such a dramatic picture

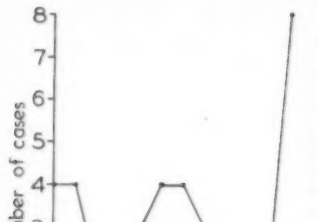

Fig. 5. Acute Intussusception—seasonal incidence. 39 cases.

that the inexperienced are misled and often fail to recognize the condition. Nevertheless, the early clinical manifestations of intussusception are almost always alarming and usually so characteristic that the correct diagnosis should be suggested at once.

It is seldom that the infant is not fat and healthy and in 80% of our cases the condition appeared 'out of the blue.' In the remainder gastro-enteritis or upper respiratory infection preceded the onset; in this connection our seasonal incidence of the condition is of interest (Fig. 5). It would appear that the minor winter peak in May to August (which corresponds to the Christmas peak in the Northern hemisphere) may be related to respiratory infections with lymphoid hyperplasia, while the peak in December to February is probably related to summer diarrhoea.

The patients in whom gastro-enteritis is complicated by acute intussusception often present a very difficult diagnostic problem. However, provided it is remembered that this might be the cause of a 'turn for the worse" the diagnosis can usually be made on the features indicated below.

Pain is always present, although it may require experience to detect it. The classical screaming with each bout of colic, although common, is not always present. Some infants simply cry more than usual, others tend to whine and whimper, while others simply blanch with the spasms.

Vomiting occurs in most of the cases. It was the most prominent symptom in 3/4ths of our cases and the *first* symptom in 20%. In babies with established obstruction the vomitus becomes profuse and dark green or brown in colour.

The bowels vary considerably but it is very rare for faecal matter to be passed after the first stool. In 10% of our patients the character of the stool was misleading tending to simulate that of gastro-enteritis, and such cases may present most difficult diagnostic problems. Blood was passed *per rectum* in 86% of our cases but in half of them it appeared only after 24 hours. It is thus obvious that it would be a grave mistake to wait for blood to appear before making the diagnosis.

In the early stages the general condition of the child is usually good, except for pallor during attacks of pain, and the abdomen is flat. Later, however, the child becomes listless and dehydrated, with abdominal distension.

A palpable abdominal mass was detected in 80% of our cases. It was found in various parts of the abdomen, most frequently in the left iliac fossa. It was always fairly large, had the characteristic sausage-shape, and never disappeared completely between contractions; and in 15% of the cases it was also palpable *per rectum*. However, failure to feel a lump should not lead to delay in diagnosis, because it is often impalpable in small-bowel intussusceptions, or may be hidden under the costal margins.

The *diagnosis* of acute intussusception must always be entertained if a healthy infant suddenly develops acute abdominal pain or vomiting. In the majority of cases the mother's description of the symptoms will be true to type—it is the very constancy of the clinical picture that suggests the diagnosis. There may be difficulty in deciding between acute intussusception and acute gastro-enteritis, and in such cases a barium-enema examination becomes imperative. Other conditions which may give rise to difficulty include worms, mesenteric adenitis, tuberculous peritonitis, Henoch's purpura, and prolapse of the rectum.

ACUTE APPENDICITIS

This is the commonest and most important 'surgical emergency' to affect the abdomen of a child. It is particularly dangerous and difficult to diagnose in children of 5 years and less. In this age-group 40-60% of the cases are already complicated on admission to hospital and the mortality is almost 10 times that of older children. (Below the age of 3 years approximately 85-90% are complicated on admission, while in neonates the diagnosis has not yet been made before perforation occurred). At the same time the disease is not as rare in this age-group as is generally thought. Various authors report that 28-35% of the cases of acute appendicitis in childhood occur in children under 5 years (about 5% occur in children of less than 3 years and about 20 neonatal cases have been reported).

TABLE III. ACUTE APPENDICITIS

Age (years)	1	2	3	4	5	6	7	8	9	10	*Total*
Uncomplicated ..	–	–	–	–	1	3	2	5	5	8	24
Peritonitis ..	–	1	2	2	2	2	2	2	3	2	18
Mass ..	–	–	2	2	2	1	1	–	–	–	8
Total ..	–	1	4	4	5	6	5	7	8	10	50

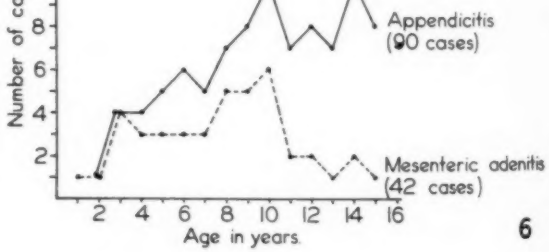

Fig. 6. Acute appendicitis and mesenteric adenitis—age incidence. 132 cases.

Table III illustrates the relative incidence of complicated and uncomplicated appendicitis at the various ages in our series and Fig. 6 indicates the increasing frequency of acute appendicitis with advancing years.

Although delay in diagnosis is an important factor in this high incidence of complicated appendicitis, the rapid progress of the disease particularly in young children plays an important part. Both these features are illustrated in Table IV, which shows that in nearly half our patients the diagnosis was delayed beyond

TABLE IV. ACUTE APPENDICITIS: DURATION OF SYMPTOMS—50 CASES

Duration	*Unruptured*	*Peritonitis*	*Mass*	*Total*
0–12 hours ..	7	—	—	7
12–24 hours ..	10	2	—	12
24–48 hours ..	5	4	—	9
48–72 hours ..	1	5	1	7
3– 4 days ..	1	1	1	3
4– 5 days ..	—	1	2	3
5– 6 days ..	—	1	3	4
6– 7 days ..	—	4	1	5
Total ..	24	18	8	50

48 hours and that in almost a quarter of those diagnosed within 48 hours the appendix had already perforated. It is therefore obvious that the diagnosis should be made within the first 24 hours if the mortality and morbidity rates are to be improved. This is possible provided the cases are seen early enough. The main features of the disease in young children which should lead to early diagnosis will be discussed under 4 headings:

1. *The Classical Symptoms*

The triad of abdominal pain, vomiting and fever in a young child must be regarded as indicative of acute appendicitis until proved otherwise. If the pain and vomiting have lasted for more than 6 hours and an enema has brought no relief, the child should be hospitalized.

It is important to realise that Murphy's sequence of pain-vomiting-fever often does not apply to the child. Any of these three symptoms may predominate and in the early stages any may be absent.

Pain is often difficult to assess, but the majority of the children indicate by their behaviour that they have a belly-ache. Localization is equally poor, and the child usually indicates the site by putting his hand to the umbilicus.

Occasionally the pain is so mild that the patient drops off to sleep and this may also happen in patients who are exhausted by advanced disease; it must be emphasized that the maxim 'Never wake a child at night to have an operation' is not dependable and may lead to serious errors. Sometimes the child may be quite bright and active between spasms of pain, which also tends to fool the unwary; it is important not to be misled by the apparent mildness of the pain. The most dangerous aspect of the pain, however, is its temporary disappearance when gangrene occurs just before perforation. If the child is seen at this stage he may even ask for food or fall asleep quietly; but other features, if looked for, will indicate that something is seriously amiss.

Vomiting is an almost universal symptom although it may be absent in the first 24 hours—it was present in 84% of our cases. It is often the most prominent symptom and in 30% of our cases it was the first symptom, which contradicts yet another maxim, viz. that pain always precedes vomiting. Indeed, vomiting may precede pain by many hours. There are no distinguishing features about the vomiting until the late stages, when it becomes faeculent.

Fever of a considerable degree may occur in young children even at an early stage. More than half of our young patients had temperatures of over 101°F, and several had temperatures of 102° up to 104°. As a rule the fever tended to be higher in the complicated cases. However, in some of the most dangerously ill patients the temperature was normal or only slightly raised. Not infrequently the temperature on admission was normal simply because of exposure to cold during transport to hospital.

The pulse rate is usually proportionately increased, but it may be comparatively slow. A steady rise is always more significant than the actual rate at a single examination.

2. *Unusual Symptoms*

Certain symptoms which are uncommon in adults tend to occur in children and may confuse the diagnosis. These are:

(*a*) *Diarrhoea*, which occured in 20% of our cases, and is particularly prone to occur in pelvic appendicitis. It may be the first and most prominent symptom (5% of our cases), especially in very young children. In these cases the stool tends to be very loose and often contains mucus, but not blood. Diarrhoea is also a symptom of the late case with a pelvic 'abcess'. Characteristically it comes on 2-3 days after the onset of pain, and consists of small amounts of mucus stained with faeces. There may be a true dysenteric type of stool, which occurs in cases of gastro-enteritis complicated by acute appendicitis. Although this type of case is rare, it very definitely

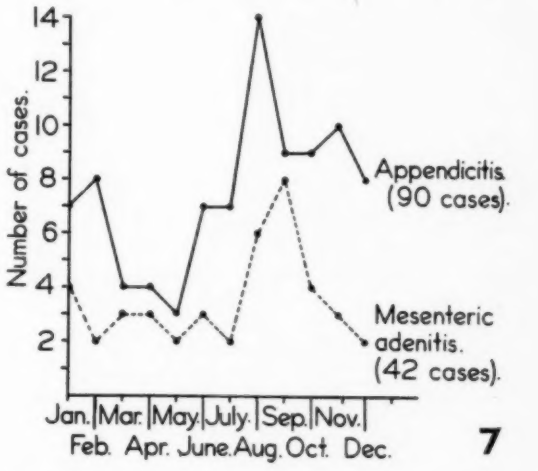

Fig. 7. Appendicitis and mesenteric adenitis—seasonal incidence. 132 cases.

does occur and may lead to great diagnostic difficulties. It should be noted that in most cases the bowels are perfectly normal prior to the attack of acute appendicitis and that constipation is a feature of late cases only.

(*b*) *Urinary symptoms*, mainly the frequent and painful passage of urine, occured in 12% of our cases; they may dominate the clinical picture. However, the urine of these cases contains only a few pus-cells and never excessive amounts, while the pain is suprapubic and not urethral.

(*c*) *Symptoms prior to the onset*. Although most of the children are in good health before the onset of the 'classical' symptoms there are exceptions, viz.:

(i) There may be a history of malaise, peevishness and refusal of food for a day or two before.

(ii) In 15% of our cases there was a history of upper respiratory infection preceding the onset of symptoms. This may have a bearing on the seasonal incidence of appendicitis (Fig. 7) and if it should be treated with antibiotics may be responsible for serious errors.

(iii) The disease may occur during the course of any of the acute infectious illnesses of childhood. One of our patients had measles and two others had chicken-pox.

3. *Misleading Aphorisms*

The fallacy of two of these has already been referred to, viz., 'Never wake a child at night to have an operation' and 'Pain always precedes vomiting'. These require no further discussion.

It has been stated that in children under the age of 6 years the appendix is never the cause of *recurrent* abdominal pain. While this may be so in the majority, there are a fair number (15% of our cases) where the history reveals a 'herald attack' of colic and vomiting 2-3 weeks before the onset of the acute attack. Furthermore, a previous history of one or more acute attacks in the past does not exclude a diagnosis of acute appendicitis.

The old teaching that the presence of headache virtually excludes acute appendicitis is equally fallacious. While it is true that headache is distinctly rare in the early stages, it is not so uncommon later in the course of the disease and sometimes it even precedes the onset of abdominal symptoms.

All these 'negative aphorisms' are of value only in that they tend to stress the rarity of certain features which, if present, should lead the clinician to re-assess the case before making a diagnosis. On the other hand, they are misleading because the inexperienced tend to accept them as 'words of wisdom' without any qualification. They are best erased from all text-books.

4. *Physical Signs*

The final diagnosis obviously rests upon objective evidence of the disease obtained from careful physical examination. In this connection, the following are significant features:

(*a*) The general appearance does not present anything characteristic and may even be misleading. Some patients tend to adopt a rather characteristic attitude—they lie either on their back with knees drawn up or curled up on their side. A flushed face, restlessness and frequent changes of postures are uncommon and should lead one to question the diagnosis.

(*b*) The tongue is almost always furred and the breath is often offensive. Some clinicians even claim that they can spot acute appendicitis 'by its smell'.

(*c*) Abdominal breathing is soon replaced by thoracic respirations. This is an important indication of 'splinting' of the abdominal muscles, but may lead the unwary into suspecting pulmonary disease.

(*d*) Tenderness is a constant sign and the most important evidence of local disease. In uncomplicated cases it may be localized to a very small area of the abdomen, and in pelvic appendicitis it is usually only slight. With spreading peritonitis the tenderness spreads and may affect the whole abdomen. The significance of local tenderness must never be underestimated and if it is indubitably present appendicitis must be strongly suspected. It must also be remembered that the appendix may occupy any part of the right side of the abdomen and may even lie in the left hypochondrium or iliac fossa; in only 50% of our cases was the tenderness at the 'classic' McBurney's point.

(*e*) Muscle guarding is a common and important sign. In the early case there is simply a difference in the tension of the two sides of the abdominal wall, best detected by light palpation with the finger tips. The area of muscle on guard may be quite small and should be sought for carefully by systematic palpation of the whole abdomen. Psoas spasm may be present in retrocaecal appendicitis, but is seldom very marked and usually considerably less than in external iliac adenitis.

(*f*) Percussion tenderness is a most valuable sign in older children but difficult to assess in those under 5 years. It serves to localize the point of maximal tenderness more accurately than palpation.

(*g*) A mass, distension or ileus all indicate advanced disease with complications. In the early case there is no distension and bowel sounds are normal, but occasionally (8% of our cases) the thickened appendix wrapped up in omentum may be palpable.

(*h*) Rectal examination, which is an essential part of the clinical examination, may reveal the presence of a pelvic mass; in 20% of our cases who had developed an abscess, the swelling could be detected *per rectum* only. On the other hand, the procedure causes so much discomfort in small children that it may be misleading as far as tenderness is concerned.

(*i*) The white-cell count shows gross variations—from 8,000 to 35,000 in our cases. In complicated cases, however, it is almost always raised above 12,500. Furthermore, if an unusual count is found, the clinical evidence should be reconsidered before a final diagnosis is made.

(*j*) The erythrocyte sedimentation rate is also variable but tends to remain low prior to rupture of the appendix. In several of our cases of acute uncomplicated appendicitis the ESR was within normal limits.

5. *Diagnosis*

Acute appendicitis must be suspected in every child

with a history of belly-ache and vomiting that has lasted for more than 6 hours. If a diagnosis cannot be made when the child is first seen, he should be examined again in an hour or two or, if this is impossible, must be hospitalized for observation.

Localized tenderness is the most useful early physical sign. If it is indubitably present and associated with muscular guarding, laparotomy is demanded.

Conditions which are responsible for most of the errors in diagnosis are acute non-specific mesenteric adenitis and gastro-enteritis. In our experience mesenteric adenitis, which occurs in the same age-group (Fig. 6) and manifests a similar seasonal incidence (Fig. 7), is most frequently mistaken for appendicitis, and in 50% of our cases laparotomy was performed because of uncertainty. It is felt that more harm can be done by 'sitting on' a case of acute appendicitis diagnosed as mesentric adenitis than by doing a laparotomy for mesentric adentitis mistaken for acute appendicitis.

Gastro-enteritis presents more difficult problems. It would be a most serious error to operate on a child suffering from gastro-enteritis, while it is also serious to treat a case of acute appendicitis as for gastro-enteritis. The former error is not frequently made but it is common practice to treat children suffering from abdominal pain and diarrhoea with antibiotics. It has already been pointed out that diarrhoea may be a prominent symptom of acute appendicitis and in such cases the antibiotics will only serve to mask the symptoms even further. The most difficult problems are presented by those patients in whom gastro-enteritis is complicated by appendicitis.

In all cases the presence of blood in the stools and excessive, turbulent peristalsis on auscultation favour a diagnosis of gastro-enteritis.

Other conditions which may cause difficulties include acute external iliac adenitis, primary peritonitis, pneumonia, and lesions of the right kidney and ureter, e.g. acute pyelitis.

CONCLUSIONS

From the above considerations it should be obvious that the *first step* towards improving the mortality rate from acute abdominal emergencies should be better education of the public. The dangers of castor oil and other 'home remedies' should be stressed, and nurses, midwives and parents should all be made aware of the potential dangers of 'upset tummies' in small children. Abel and Allen have demonstrated that a great deal can be done by public education, and it is the duty of general practitioners to pursue this most energetically.

Secondly, practising doctors must be made aware that surgical emergencies may occur at all ages from birth onwards, e.g.:

Age	*Surgical Emergency*
Days	Congenital intestinal obstructions.
Weeks	Pyloric stenosis.
Early months	Irreducible hernia.
Later months	Acute intussusception.
Years	Acute appendicitis

It requires experience to teach us how to handle a small child, but a great deal can be done by teaching students the importance of a carefully taken history and a painstaking, gentle and unhurried physical examination. Various subterfuges, such as examining the child under the bedclothes or while he is sitting on his mother's lap, are helpful but the final diagnosis still depends on attention to detail, adequate knowledge and careful interpretation of physical signs.

Thirdly, the value of repeated examinations and close observations cannot be over-emphasized. When seen for the first time the child may present a most puzzling clinical picture and the exact diagnosis is often in doubt. It is the doctor's duty not to prescribe 'shot gun' antibiotic therapy in such cases, but to examine the child again after a couple of hours and yet again if there is still doubt. If his other work does not allow of such 'unceasing vigilance', the doctor must arrange for the child's admission to hospital, where he can be kept under close observation and examined frequently and repeatedly. No hospital authority has the right to refuse admission to a vomiting infant or a child with a belly-ache and, even if only one out of every ten admissions turns out to be a true surgical emergency, all the admissions are more than justified.

Lastly, there is the burning question, 'Shall we operate or not?'. This is a problem which should concern the surgeon rather than the practitioner, and in hospital practice, where all facilities are at hand and 'pressure' from the relations and family doctor usually does not exist, the decision is mostly not so difficult. In private practice, on the other hand, there is often a certain amount of bias which renders accurate judgment most difficult; some parents may violently oppose surgical intervention while others may be over-enthusiastic about an operation, and the same applies to many practitioners and paediatricians. Only too often the easiest way out of the difficulty is to 'look and see' rather than to 'wait and see'. In this connection it must be pointed out that every operation, no matter how minor, carries a certain mortality and morbidity and that unnecessary operations cost lives. On the other hand, it may be equally disastrous to 'wait and see' in a case that requires an urgent operation.

In Table V are listed a number of conditions which have brought children to our surgical wards with a provisional diagnosis of acute surgical emergencies. Some of these have already been discussed in relation to acute intussusception and acute appendicitis. Many of them may give rise to great difficulties in diagnosis and their varied nature serves to emphasize the im-

TABLE V. CONDITIONS SIMULATING ACUTE ABDOMINAL EMERGENCIES

A *Extra-Abdominal Diseases with Associated Abdominal Symptoms*	*B* *Abdominal—Operation Harmful*	*C* *Abdominal—Laparotomy Justifiable if Doubt Exists*
Infections of throat and ears.	Gastro-enteritis.	Mesenteric adenitis.
Pleurisy and pneumonia.	Intestinal colic.	Iliac adenitis.
Osteitis of spine and pelvis.	Pyelitis.	Primary peritonitis.
Meningitis.	Ureteric colic.	Regional enteritis.
Infectious fevers.	Infective hepatitis.	Acute pancreatitis.
Rheumatic fevers.	Typhoid.	
Poliomyelitis.	Abdominal allergy.	
Bornholm disease.	Worms.	
	Faecal impaction.	

portance of a thorough and complete physical examination in every case that presents as an 'acute abdomen'. This should always lead to the correct diagnosis of extra-abdominal diseases with associated abdominal symptoms (A) and laparotomy in such cases would be unpardonable.

Under (B) are listed abdominal conditions which may simulate surgical emergencies very closely. These conditions must always be excluded in a suspected case because laparotomy would be most harmful and, if a final decision cannot be made, the wisest policy would be to 'wait and see'.

Under (C) are listed other abdominal conditions which often mimic surgical emergencies so closely that a diagnosis is impossible. Although operative intervention is not the correct treatment for these conditions, a diagnostic laparotomy is often the only method of excluding a more serious surgical emergency, and hence if there is doubt the best policy would be to 'look and see'. Several of these conditions might require a later operation, and laparotomy would not be as harmful as in those mentioned under (B).

SUMMARY

1. It is pointed out that the mortality of acute abdominal emergencies in children still leaves much room for improvement.

2. The excessive mortality is attributed to delay in diagnosis and the causes of such delay are discussed.

3. Common surgical emergencies are discussed and their features illustrated by an analysis of 314 cases treated over a period of 2 years.

4. The diagnostic features of neonatal intestinal obstructions, pyloric stenosis, and irreducible hernia, are briefly discussed.

5. The clinical features and diagnosis of acute intussusception and acute appendicitis are dealt with in some detail.

6. It is concluded that earlier diagnosis could be made possible by (*a*) education of the public, (*b*) improved clinical training of students, (*c*) increasing vigilance with repeated examination of all suspected cases, (*d*) hospitalization if there is any doubt, (*e*) timely laparotomy in selected cases.

I wish to thank Dr. N. H. G. Cloete, Superintendent of the Groote Schuur Hospital, and Dr. J. Mostert, Superintendent of the Red Cross War Memorial Children's Hospital, for permission to publish these observations. My thanks are due to Mr. A. Katz, who has assisted me in the Children's Department throughout, to my registrars and house surgeons, and to the ward sisters and their nursing staff.

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SURGICAL CLOSURE OF AURICULAR SEPTAL DEFECT

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Durban

The decade since the war has witnessed startling developments in surgery of the heart. Extracardiac operations for the treatment of patent ductus arteriosus and coarctation of the aorta are now accepted and everyday procedures. The construction of a systemo-pulmonary anastomosis of the Blalock or Potts type in the treatment of the tetralogy of Fallot is performed in many units in preference to the direct procedures developed by Brock. Pericardectomy for the relief of constrictive pericarditis is well established. Operative procedures upon the heart itself embrace the relief of stenosis of all the valves; mitral valvotomy is one of the major triumphs of surgery; pulmonary and tricuspid valvotomy pose no real problems for the surgeon; and even aortic valvotomy, although a much more hazardous procedure, is being effectively tackled. But the problems presented by the surgical treatment of valvular incompetence and coronary insufficiency are far from being solved.

The problem of closing abnormal communications between the two sides of the heart has appeared very formidable. It had seemed that open cardiotomy with the aid either of an artificial circulation or of hypothermia would be required. These techniques have in fact been employed in the treatment of these conditions, but they are still fraught with grave risks. Safe repair of defects between the two ventricles is still a vision for the future, but the repair of defects between the two auricles is much more hopeful and many relatively safe procedures have now been devised without recourse

to the risks of open cardiotomy. Much ingenuity has been displayed by the various workers in this field. A remarkably simple technique has been described by Senning[1] and Parantela,[2, 3] which indicates that what has been a major problem may turn out to be capable of a fairly straightforward solution. In this article I describe a case successfully treated by their technique.

EMBRYOLOGY AND MORPHOLOGY

A short review of the embryological development of the atrial septum is necessary for the understanding of the morphology of the various septal defects encountered. Briefly, the single primitive atrium is first divided by the septum primum, which grows down and forward towards the developing ventricular septum. However,

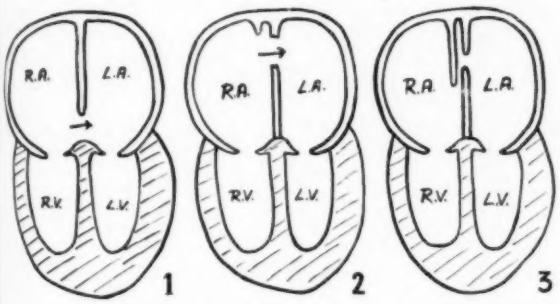


Fig. 1. Down-growth of septum primum dividing primitive auricle. Arrow indicates ostium primum.
Fig. 2. Upper portion of septum primum has broken down to form ostium secundum (arrow) appearing to right of septum primum.
Fig. 3. Ostium secundum now closed by downgrowth of septum secundum.

a communication—the ostium primum—remains for a time just above the ventricular septum but eventually closes (Fig. 1). The upper part of the septum primum gradually breaks down (Fig. 2), but this opening—the ostium secundum—is closed by the development of the septum secundum, which grows downward to the right of the original septum, the free edge eventually forming the annulus ovalis of the foramen ovale (Fig. 3). The overlapping septa explain the valvular action of a persistent foramen ovale, which is physiologically closed by the greater pressure in the left auricle.

One can readily appreciate how imperfect development may result in a persistent ostium primum, or persistent ostium secundum, or various combinations of the two. These defects may be relatively small or may amount to a virtual absence of the septum. In practice it is found that most defects are situated postero-superiorly and are, in fact, persistent ostia secunda. A persistent ostium primum is frequently combined with congenital defects of the mitral and tricuspid valves, and the fact that the bundle of His is very close to the inferior margin renders effective and complete surgical repair rather hazardous. Fortunately this does not apply to the repair of the commoner type (approximately 90%) which is situated well away from the bundle of His.

PATHOLOGICAL EFFECTS

The pathological effects of an auricular septal defect can readily be appreciated. One must exclude cases complicated by tricuspid or pulmonary stenosis or atresia, in which the defect is life-saving and allows a right-to-left shunt to persist. In uncomplicated cases a left-to-right shunt occurs because of the higher pressure in the left auricle. This results in an increased circulation through the lungs and the pulmonary flow may be several times greater than the systemic flow and secondary pulmonary hypertension and pulmonary artery atheroma may ensue. The right auricle and the right ventricle enlarge, the pulmonary artery becomes prominent and the lungs become plethoric. As the shunt is from left to right cyanosis is absent. The right side of the heart becomes subject to considerable strain. Effort dyspnoea and orthopnoea is usually present and eventually congestive failure may supervene. Sub-acute bacterial endocarditis as a complication is fortunately almost unknown. The degree of disability varies very considerably but in most cases is quite marked and the eventual prognosis is poor. Some cases survive to middle age. Frequently difficulty only commences in the 4th decade.

DIAGNOSIS

A typical case will manifest a systolic murmur over the base of the heart. This is thought to be due to the increased flow in the pulmonary artery and not to the flow through the shunt. The second pulmonary sound is usually widely split and frequently accentuated. Evidence of right ventricular hypertrophy can usually be obtained by palpation. The radiological appearances are fairly characteristic and usually indicate enlargement of the right auricle and ventricle and characteristic prominence of the pulmonary artery, which usually displays marked pulsation or 'hilar dance'. Pulmonary plethora is usually present. These changes may be slight or very gross. The electrocardiogram usually displays evidence of right heart strain and very frequently evidence of an incomplete right bundle-branch block (95%).[4] This is thought to be due to the relative hypertrophy or dilatation of the right ventricle muscle, causing more widespread conduction of the electrical impulse. Cardiac catheterization should confirm the diagnosis by demonstrating an increase in oxygen saturation of blood in the right ventricle as compared with caval blood. However, one must beware of being misled, for aberrant entry of a pulmonary vein into the right auricle will give a similar finding. The catheter may pass from the right auricle into the left auricle but this is not confirmatory of an atrial septal defect, for the catheter may pass through a persistent foramen ovale with a valvular action which is yet able to prevent a shunt in the reverse direction. The role of angiocardiography in the diagnosis of atrial septal defect is limited, but it may be indicated and may prove helpful if the diagnosis is in doubt.[5] In most cases careful assessment of the various physical signs and investigations will enable the diagnosis to be confidently made. The persistent ostium-primum type of defect should

be suspected if there is evidence of mitral or tricuspid incompetence.

SURGICAL CLOSURE

Numerous techniques for surgical closure have been devised. If open cardiotomy, under hypothermia or with the aid of an artificial circulation, were a safe procedure, direct suture of the defect under vision would be the recommended procedure. However, this is not yet attainable, although several series of operations on these lines have been reported.[6] I do not propose to discuss all the closed techniques which have been suggested. They are legion. Amongst the methods that have been attempted are sutures passing from front to back, through the auricles in the line of the atrial septum,[7] and the indentation and suture of the redundant lateral wall of the right auricle to the margin of the defect,[8] thus producing a doughnut-shaped auricle (Figs. 4 and 5). These methods cause considerable

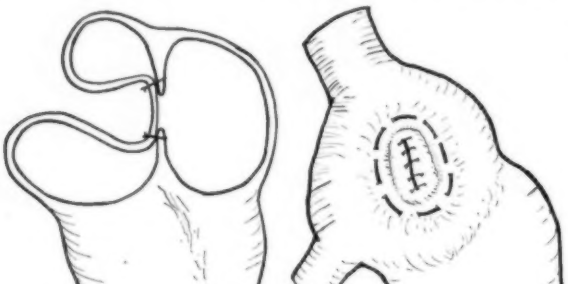


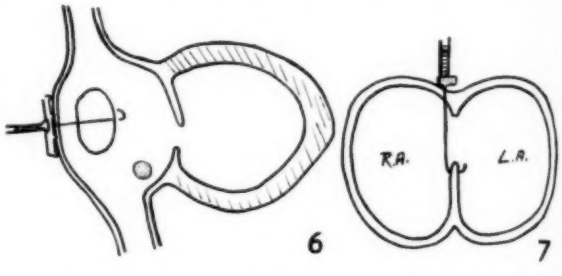
Fig. 4. Lateral wall of right auricle indented and sutured to margins of atrial septal defect.
Fig. 5. Doughnut-shaped auricle produced by atrioseptopexy.

deformity of the right auricle although good results are claimed by the latter method,[8] and it is stated that an adequate lumen exists in front of and behind the sutured portion. Gross has developed an ingenious method of operating through a rubber well-sutured to an incision in the wall of the right auricle and which when filled with blood offers an entry to the interior of the heart without affecting the circulation.[9] An objection to this technique, which also applies to closure by open cardiotomy, is the danger of the stitches tearing out of the edges of the defect because relaxation and lack of tension have not been obtained.[10]

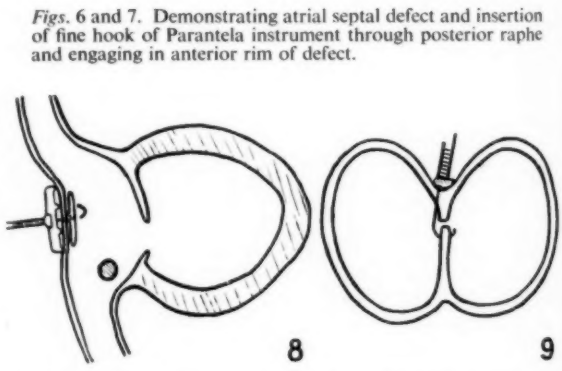
Numerous other methods have also been devised and some have been applied clinically,[11, 12] but the most fruitful line of approach appears to be one where the junction between the auricles posteriorly is mobilized and this posterior wall or raphe is displaced forward and sutured to the anterior margin of the defect. This causes the minimal amount of distortion of the auricles and avoids undue tension upon the sutures. This especially applies since most of the defects are situated in the posterior portion of the septum. Several applications of this principle have been described,[13] but the simplest and most effective has been evolved by Senning[1] and Parantela.[2, 3]

SENNING AND PARANTELA'S OPERATION

Parantela has devised a rather ingenious instrument which facilitates this technique.* A right thoracotomy is performed, the pericardium is widely opened anterior to the phrenic nerve, and the right auricle and the superior and inferior venae cavae are displayed. The left forefinger is introduced through an incision in the appendix of the right auricle and the defect in the auricular septum is palpated. The superior and inferior vena-caval openings and the coronary sinus opening are located and the tricuspid valve is palpated. A finger can be passed through the defect into the left auricle and the pulmonary vein openings should be palpated to ensure that they enter normally into the left auricle. This is important, since aberrant entry into the right auricle can be corrected. The mitral valve is then palpated to ensure that mitral stenosis is not present (Lutembacher's syndrome). The finger is then withdrawn and an appendicular clamp applied to the appendix, and the following dissection then performed (Blalock-Sondergaard). The right pulmonary veins are closely adherent to the back of the right auricle, where they come in contact in process of passing to the back of the left auricle. By dissection the plane between these veins and the back of the right auricle is entered and, by continuing the dissection to the left, one can display the site of junction of the two auricles posteriorly. The superior and inferior venae cavae are adequately mobilized. The left forefinger is again introduced through the appendix of the right auricle and it will be found that the posterior wall at the raphe can be displaced forward to the anterior rim of the defect if adequate mobilization has been obtained. The special hook of the instrument is then introduced through the posterior raphe and hooked into the anterior margin of the defect, being guided by the finger inside the heart (Figs. 6 and 7). The cross-bar of the instrument



Figs. 6 and 7. Demonstrating atrial septal defect and insertion of fine hook of Parantela instrument through posterior raphe and engaging in anterior rim of defect.



Figs. 8 and 9. Posterior wall displaced forward to anterior rim of septal defect and position maintained by Parantela instrument.

* Heljestrand-Parantela instrument, manufactured by Heljestrand, Eskilstuna, Sweden.

is then slowly moved forward until the posterior raphe comes in contact with the anterior margin (Figs. 8 and 9). If the defect is large, one or both of the additional hooks, which can be incorporated in the instrument, will need to be similarly inserted. The final position is maintained by a ratchet. It will now be found that the defect has been temporarily closed with minimal distortion of the auricles. External sutures can now be simply inserted from the back of the heart around the cross-bar of the instrument, as the anterior lip of the defect has in fact been brought in direct contact with the posterior raphe. The finger in the heart assists in the placement of these sutures, which are tied by the assistant, and the instrument and the finger are then withdrawn.

This technique was adopted in the case here reported and proved to be very straightforward and satisfying. There was almost no loss of blood and the heart appeared relatively unaffected by the manipulations, apart from an occasional extra-systole. This method is applicable to most atrial septal defects but in the ostium-primum type it may be wisest not to place any sutures in the lower end for fear of damaging the bundle of His. If aberrant insertion of a pulmonary vein into the right auricle is present, then one can displace the right auricular wall to the right of the aberrant vein forward to the anterior rim of the defect, and the aberrant vein will now enter to the left of the reconstructed septum and thus into the left auricle. If the aberrant vein enters the superior vena cava the same principle is adopted.

CASE RECORD

Mrs. K.A., aged 32 years, referred by Dr. H. Schaffer and Dr. J. Rossiter with the diagnosis of auricular septal defect.

Patient was quite well until 6 years ago. Since then there had been progressive dyspnoea. For the past year she became dyspnoeic after very moderate exertion. She was unable to climb stairs. She preferred sleeping with several pillows. She was originally told that she had mitral stenosis.

On examination (27 September 1955). The patient was somewhat obese, with a very limited exercise tolerance. There was no evidence of congestive cardiac failure. A marked right ventricular heave could be palpated. There was a blowing systolic murmur over the pulmonary area and a widely split and accentuated second pulmonary sound.

On screening there was seen to be moderate cardiac enlargement, which appeared to be confined to the right auricle and the right ventricle. The left auricle revealed no signs of enlargement. The pulmonary-artery segment was markedly prominent and convex, and displayed vigorous pulsation or 'hilar dance'. The vascular shadows in both lungs were very prominent.

The electrocardiogram showed evidence of right heart strain and broadened QRS complexes (0·12 seconds) which were considered to indicate partial right bundle-branch block (Fig. 10).

Cardiac catheterization was performed on 14 October 1955 (Dr. T. G. Armstrong). The findings were as follows: Pressure in right auricle 6/0 mm. Hg, in right ventricle 30/0 mm. Hg, in pulmonary artery 30/10 mm. Hg.

The following oxygen saturation figures were obtained: Peripheral arterial 92·5%, superior vena cava 67%, right auricle 78%, right ventricle 80%, pulmonary artery 80%.

The diagnosis of atrial septal defect appeared very definite. However, we were not convinced that the severity of the patient's symptoms justified operative intervention and she was observed for several months. Her dyspnoea became progressively worse and it was eventually decided to repair the defect surgically.

The operation was performed on 29 February 1956 under general anaesthesia, administered by Dr. H. Grant Whyte and with ECG monitoring (Dr. D. Pittaway). The defect in the auricular septum was situated postero-superiorly and was considered to be a persistent ostium secundum. It was roughly circular in shape and was estimated to measure 3 × 3 cm. The mitral and tricuspid valves were normal and there was no evidence of aberrant insertion of the pulmonary veins into the right auricle. The method of closure employed was that which has been described above, and was performed quite uneventfully. Three sutures were required. There was no loss of blood and the heart tolerated the manipulations very well, apart from an occasional extra-systole. Post-operative convalescence was uneventful and patient was discharged home on 14 March 1956.

She has revealed marked clinical improvement and is no longer dyspnoeic on exertion. She has no orthopnoea. X-ray screening does not reveal any change in the contour of the heart. The pulmonary-artery segment is still prominent and convex but the 'hilar dance' has ceased. Pulmonary plethora is no longer present. The second pulmonary sound is still split but does not appear to be as accentuated as it was previously.

Electrocardiogram (1 May 1956) reveals a definite change in the length of the QRS complexes, which now measure 0·08 sec. (Fig. 10). The full significance of these changes is uncertain they appear sufficiently definite to suggest that there is now less strain on the right side of the heart.

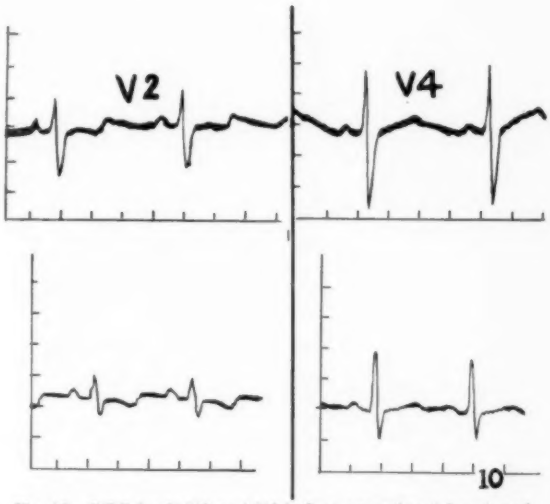


Fig. 10. ECG leads V2 and V4 before operation (above) and 2 months after closure of atrial septal defect (below).

SUMMARY

A case of successful closure of an auricular septal defect is presented. A relatively simple and effective technique is suggested and the main principles described.

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OESOPHAGOPLASTY BY MODIFIED MES METHOD

CASE REPORT

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Mrs. A.D., a European aged 21, was admitted to Groote Schuur Hospital on 18 February 1955 complaining of dysphagia. She had swallowed caustic soda—a teaspoonful in half a cup of water—on 8 November 1954. Two weeks later she began having difficulty with swallowing, and this had been progressive.

On admission she was able to swallow fluids but not solids. Examination showed her to be in good general condition, though she had lost a little weight. A barium swallow showed a long severe stricture of the oesophagus beginning at the upper thoracic inlet and extending to the diaphragm. She was then transferred to the Ear, Nose and Throat wards.

Left: *Fig.* 1. Shows the extent of this formidable stricture considered radiologically to begin at D4. In fact the stricture began about 2 inches higher, and extended to the diaphragm.

Right. *Fig.* 3. Barium passing through the site of anastomosis in the neck one month after the anastomosis had been made.

From 24 February to 29 June dilatations of the oesophagus were performed, with varying success. On at least two occasions she showed evidence of mediastinitis, which was controlled with antibiotics. It was agreed that dilatation offered no chance of permanent cure and it was decided to proceed with the Mes manoeuvre.

On 29 June the abdomen was opened through a left paramedian incision and the greater curvature of the stomach freed from the greater omentum in the manner described by Mes, with careful preservation of the gastro-epiploic artery. The stomach was then buttonholed through both surfaces about 1½ inches proximal to the pylorus, and a pair of long clamps applied parallel to the greater curvative and about 2 inches from it. The stomach was then divided between the clamps, leaving the smaller inferior part attached at the pyloric end. The open surface of the stomach was then closed in two layers, while the lower portion was stretched to its fullest extent and converted into a tube with interrupted linen thread sutures in two layers. The stomach now presented the appearance of a Y and great care was taken not to narrow the lumen at the junction of the limbs of the Y.

Before the abdomen was closed, the (left) thorax was opened in the 5th interspace and the tube of stomach introduced into the thorax through a small opening in the diaphragm alongside the oesophageal hiatus. A jejunostomy tube was placed in the upper jejunum and the abdomen closed. The tube of stomach was tacked to the posterior mediastinal pleura with a few sutures. A 3-inch incision was then made in the neck, along the anterior border of the left sternomastoid from the jugular notch upwards, and by blunt dissection a passage was made between thorax and neck, anterior to the great vessels, and the tube of stomach was brought out in the neck and tacked to the skin. The thorax was then closed, with under-water drainage.

The patient tolerated the operation very well and was maintained by jejunostomy feeds. On 3 July the wound in the neck was reopened and the tube of stomach mobilized from its bed. The inferior thyroid artery was identified and ligated and the thyroid gland mobilized towards the mid-line to permit easier identification and mobilization of the oesophagus. The latter was mobilized with some difficulty, probably ascribable to previous peri-oesophagitis due to the caustic soda. When the oesophagus was opened in the root of the neck the wall was found to be densely

Fig. 2. Thick barium had been poured down the tube and the patient had swallowed a small quantity of thin barium. The picture shows the meeting of these two streams at the Y in the stomach—before the anastomosis in the neck had been completed.

scarred and the stricture to commence considerably above the level indicated by the X-ray—in fact tissue approaching normal was found only at the level of the thyroid cartilage; the base of the tongue was easily palpable through this opening. A satisfactory anastomosis was contrived at this level with two rows of interrupted thread sutures. There was no tension on the anastomosis. The distal oesophagus was ligated with strong chromic catgut, the mucous membrane above the ligature thoroughly destroyed with the caustery, and the stump pushed back into the mediastinum. The wound was closed and a glove drain left.

This operation was tolerated very well. After 72 hours the patient was allowed to swallow fluids. On the 5th day a fistula developed. This had closed spontaneously 6 days later and the patient was taking liquids with ease. On 27 July the jejunostomy tube was removed and semi-solids permitted—and taken well. At the end of a month she was on normal diet.

The patient returned on 30 September complaining once more of difficulty in swallowing and was found to have a soft stricture at the anastomosis. This was very easily dilated to the maximum dilator size.

The patient has a certain amount of regurgitation if she lies prone directly after a meal. This is a disadvantage and it is too early as yet to know whether there will later be trouble with stricture formation.

COMMENT

This case is described in an endeavour to draw attention to the technique evolved by Mes. He demonstrated several very successful cases at the 1948 South African Medical Congress in Pretoria, when the late T. Lindsay

Fig. 4. Shows the length of the constructed tube. The lowest forceps points to the pylorus. The second is at the Y in the stomach. The metal strip, which has been outlined for the sake of clarity, is 9 inches in length.

Sandes described the method as a significant and well-thought-out step in the treatment of these difficult cases.

The technique used in this case differs slightly from that of Mes in that the tube was brought through the thorax and not ante-thoracically. Mes also used an ingenious staged method for making the gastro-oesophageal closure in the neck. The use of the antibiotics now available renders this unnecessary. The striking advantage in his method is the ease with which a very long tube with excellent blood supply is available to bridge the gap between the diaphragm and the neck. In the case here described the tube reached easily beyond the level of the zygoma and could have been made even longer by using Mes' manoeuvre of taking in the greater curve round the cardia as well. Moreover there was definite arterial bleeding at the very end of the tube and its colour was excellent throughout its length.

Since the first case was operated upon by the Mes method, Prof. J. H. Louw has used the method on several children with a combined abdomino-thoracic incision which has proved a great improvement. In addition a duodenal tube has been introduced down the new 'tube of stomach' with its tip beyond the pylorus and the patient fed *via* this tube. This replaces the jejunostomy used in the first case, and is obviously better from every point of view.

The method has distinct advantages over the standard oesophago-gastric anastomosis. It obviates the discomfort after meals caused by the distension of the intra-thoracic stomach. The incidence of oesophagitis caused by acid regurgitation should theoretically be much less also, for the tube can be assumed to the largely 'vagotomized'. The method should be applicable in malignant disease of the oesophagus both as a palliative and as a curative procedure.

My thanks are due to Dr. F. D. du T. Van Zyl who placed this case in my care and to Mr. B. T. Todt who was responsible for the photographs.

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POSTSCRIPT

17 August 1956. The patient writes to say she has no difficulty whatsoever in swallowing.

FRIEDREICH'S ATAXY WITH AN INTERESTING FAMILY TREE*

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Friedreich's ataxy is a heredofamilial disease of the nervous system characterized pathologically by a primary neuronic degeneration of certain conducting tracts of the spinal cord and clinically by a progressive incoordination of voluntary movement. Both sexes are affected, males slightly more often than females, and several siblings in each generation may manifest the disease. The transmission may be direct or indirect, but is usually through unaffected members of tainted families, male or female, since victims of the disease seldom procreate. Some maintain that transmission cannot be explained merely on the Mendelian theory of domirant and recessive genes and regard this as an example of the so-called Gower's abiotrophy—a conception which assumes an inborn defect of vitality in certain homologous units of the nervous system leading to their premature degeneration.

The spinal cord is wasted throughout its length but especially in the thoracic segments. There is extensive degeneration of the conducting tracts of the cord, most marked in the columns of Gall, the posterior spino-cerebellar tracts and the pyramidal tracts. The columns of Burdach and the anterior spino-cerebeller tracts are similarly but less severely affected. The nerve cells of the cerebellum and brain-stem may be unaffected.

The onset of the illness is usually between the ages of 5 and 15 years and in any one family the onset is usually fairly constant.

The earliest symptom is awkwardness of gait. The child trips and falls about excessively and is unable to play games as actively as other children. Gradually the characteristic gait develops. The child walks with a wide base and the feet are flung out clumsily, raised too high off the ground and brought down with a stamp. When standing the body sways to and fro and Romberg's sign is present. Gradually a similar order of disturbances appears in the upper limbs. The movements of fingers become slow and laboured, feeding becomes clumsy, dressing takes longer than formerly. All delicate movements normally carried out subconsciously require intense voluntary effort for their completion. Function is further disturbed by the presence of a coarse intention tremor which appears in connection with all voluntary movements.

Speech shares the incoordination of the other motor activities; it becomes slow, slurred, hesitant and explosive—sometimes rising to a shout and then falling away to a whisper. As the disease advances the dysarthria may become so extreme as to make the patient's conversation unintelligible.

When the patient is standing or sitting a nodding tremor of the head and oscillating movements of the trunk are usually present. He is not aware of sensory disturbances, the special senses are not involved, and sphincteric control remains unimpaired.

Mental symptoms are usually not conspicuous, but some of the patients are of poor mentality from the first while others show a tendency to severe mental degeneration in the later stages of the disease.

As the disease advances and the pyramidal degeneration increases power of movement is gradually lost and paralysis may be universal.

Contractures of the lower extremities are the rule, producing the characteristic deformity of pes cavus. Also present is the deformity of the hand with hyperextension of the terminal phalanges analogous to talipes in the foot. Spinal curvature is very common, consisting of a scoliosis of the dorsal region with some kyphosis and with a compensatory reverse lumbar curve.

Ocular movements are usually intact, apart from fine, regular nystagmus or coarse, irregular jerkings, chiefly upon lateral deviation. Strabismus, diplopia, ptosis and optic atrophy may be present.

Tendon reflexes are reduced and usually absent, the abdominal reflexes lost, and the plantar responses extensor in type.

The cerebrospinal fluid presents no abnormality but occasionally, and especially in early cases, an excess of protein and cells may be found, and in such cases an unexpectedly strong Lange's colloidal-gold reaction, usually of a paretic type, may be present.

The diagnosis of the condition is usually not difficult. If progressive paraplegia develops towards the end of the first decade of life in a child with a family history of similar trouble, if knee jerks are absent whilst the big toes are permanently erect with an extensor Babinski, and if there is neither atrophy nor pseudo-hypertrophy of the muscles, the diagnosis is in all probability Friedreich's ataxy.

* A paper read at a meeting of the Cape Eastern Branch of the Medical Association of South Africa, Grahamstown, April 1956.

The course of the disease is usually slowly but relentlessly progressive. Voluntary movements become more and more ataxic, walking becomes impossible, and the patient may even become bedridden and utterly helpless at as early an age as 30 years and succumb to an intermittent infection. In some cases—usually by families—it seems to have no tendency to shorten life.

THE AFFECTED FAMILY

During December 1955, 7 patients, consisting of 5 sisters and their 2 brothers (marked A in Fig. 1), were admitted to the Fort England Hospital, Grahamstown. They were all suffering from heredofamilial ataxy conforming to the same pattern, the clinical picture varying only in the degree of the severity of the various symptoms. They were severely crippled, some were quite helpless, and all need supervision and nursing care for the rest of their existence. The ages ranged from 33 years to 52 years.

I was also able to visit and examine the family (marked B in Fig. 1) with 4 sons similarly afflicted with the disease. These also conform to the same pattern and the variation is in degree only.

All were normal infants, attended school and developed to the satisfaction of the parents both mentally and physically. Before the end of the first decade the disease had clearly manifested itself and soon all the physically crippling effects described above were in evidence. Not only did intellectual development cease but retrogression actually set in, so that all now suffer from defective mental development to a degree of imbecility.

Once the initial physical examinations had been completed further interest centred on the family history and as can be observed from the accompanying charts (Figs. 1 and 2) this provided a most tragic, nevertheless clinically absorbing story. It is clear that the first and third wives were tainted, for they were related (though the exact degree of relationship could not be established) and a brother of the first wife had descendants suffering from the disease (see Fig. 2).

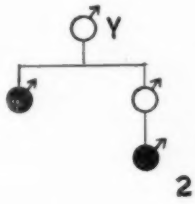

Fig. 2. Descendants of Y, a brother of wife I (see Fig. 1). Cases of Friedreich's ataxy shown in black.

The charting of the family tree has its practical application in the problems of genetical prognosis. The offspring of the 1st and 3rd wives will be tainted whereas those of the 2nd wife will be unaffected, and this has formed the basis of one's advice.

After explaining the pathology, prognosis, etc. of the condition and divulging the cold heredofamilial facts conveyed by the chart one has to leave the ultimate decision of procreation to the individual concerned.

I wish to thank Prof. I. R. Vermooten, Commissioner for Mental Hygiene, for his kind permission to publish this paper and also those members of the family who so patiently attended to all the enquiries.

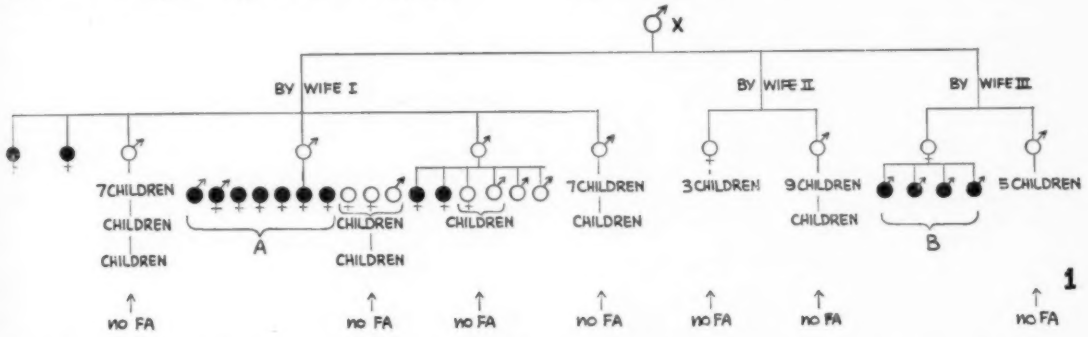

Fig. 1. Descendants of X ('untainted') and his 3 wives (I and III—'tainted'—were related; II was 'untainted'). Cases of Friedreich's ataxy (FA) shown in black.

NATIONAL CANCER ASSOCIATION : ANNUAL MEETING

ADDRESS BY DR. LEWIS S. ROBERTSON

At the annual general meeting of the National Cancer Association of South Africa, which was held in Johannesburg on 24 August 1956, the President, Dr. Lewis S. Robertson moved the adoption of the report and audited accounts for 1955. He said:

The Central Appeal Committee, under the chairmanship of Dr. H. J. van Eck, with the help of 381 local committees, had, up to 31 December 1955, raised over £325,000 for the National Cancer Appeal Fund. The Fund was administered by a Board of Trustees, without whose consent no expenditure could be made from the Fund by the National Cancer Association or otherwise. Separate accounts were kept by the Association and during 1955, owing to a refund of £3,160 received from the Board of Trustees and receipts from various sources, the Association's accumulated funds (apart from the Lady Cade Memorial Fellowship Fund and the King George V Memorial Fund) increased by £4,161 to £9,271.

The Association had, at the request of the *Johannesburg Star*, administered the money collected by that newspaper to enable a patient (Petronella) to be taken to England to consult a specialist and accept such treatment as he advised, but the Association had had no other connexion with the collection of this particular fund.

RESEARCH FELLOWSHIP

In 1951 at the request of the National Cancer Association the South African Institute for Medical Research instituted a Fellowship in Cancer Research, and Dr. A. G. Oettlé was appointed, the National Cancer Association assuming the financial responsibility. Dr. Oettlé had undertaken the following researches:

1. To investigate the effects of environment on cancer incidence, a 3-year survey of the Johannesburg Bantu was undertaken in collaboration with Dr. J. Higginson, and has just been completed. This provided accurate figures for the frequency of the commoner types of cancer in terms of new cases per 100,000 of each sex and age group in the Bantu population. Profound differences from White cancer rates have been observed. The experience gained in this survey furthermore has assisted in the design of other rate studies under the auspices of the National Cancer Association in other parts of the country.

2. For the investigation of rarer cancers, a cancer ratio study has been commenced, making use of over 15,000 cancers diagnosed in 5 years at the histopathological department of the Institute. This is expected to reveal further differences in cancers affecting the different races.

3. The discovery at this Cancer Unit of a remarkable susceptibility of a South African rodent, *Mastomys*, to spontaneous

carcinoma of the glandular stomach has attracted wide interest. Experimental studies of gastric adenosarcoma have been greatly hampered by the lack of suitable experimental animals.

SUPER-VOLTAGE RADIOTHERAPY

Dr. Robertson went on to say: The publicity given in recent months to an unfortunate child and the impression created that she was sent to Britain particularly for radio-active cobalt bomb treatment, precipitated considerable discussion in the medical and lay press why such treatment was not available in South Africa. A statement was made that there were 20-25 similar cases in South Africa who could have been cured by this treatment; but the statement has not been verified. Eventually the child did not receive any form of radiation, the treatment being entirely surgical.

While it is true that Sir Stanford Cade, to whom the child was sent, has been combining super-voltage treatment with surgery, there is no question of radiation alone from whatever source, curing more than a very small percentage of cases or even prolonging their lives to any extent.

Ever since Roentgen's discovery some 60 years ago there has been a striving for higher-voltage machines, because as the penetration of the rays increased with the voltage, deeper-seated conditions would come within the field of adequate radiation. A one-million-volt X-ray machine installed in St. Bartholomew's Hospital in London came into operation in 1938. Since the War, various super-voltage machines have been constructed and the first linear accelerator was erected at the Postgraduate Hospital in London in 1953. There are in Britain today approximately 6 linear accelerator machines ranging from 4 to 15 million volts, and there are many large hospitals in Britain without super-voltage machines.

The use of machines of very high voltage requires even greater clinical and technical skills than conventional radiotherapy in determining the dosage given and avoiding dangerous reactions. South Africa at present has not the clinical and technical resources to make general use of such machines. It has taken some years to obtain the services of physicists for our main provincial hospitals. Groote Schuur Hospital, Cape Town, lost the physicist it obtained from the C.S.I.R. and has only recently appointed a physicist to fill the vacancy. Johannesburg Hospital and the Natal Hospital Service have also each recently acquired a physicist. One physicist in a large hospital to do all the routine work of the department as well as the work connected with high-voltage and rotation therapy, is inadequate. At the Royal Marsden Hospital, London, for instance, there are about 20 physicists employed.

On the clinical side, although at some provincial hospitals there are radiotherapists with many years experience, at others none with great experience in radiotherapy and particularly of super-voltage therapy are available.

Dr. Robertson then said that because high-voltage machines had been in operation for so short a time there was considerable doubt which type was the best, and which machine should be universally used. In spite of the vast development of the X-ray field during the past 50 years, particularly in the last 20 years, death rates from cancer had continued to rise. There were many cancers which did not respond to X-ray treatment at all, no matter how large the machine. A cancer of the lung, or the stomach, or the large bowel could not be cured with X-ray beams at any voltages; it was obvious that there were other factors besides the radiation which took part in the successful eradication of a cancer. There were radiosensitive cancers that responded to radiation and other cancers that were completely non-radiosensitive. The vast majority of cancers still had to be treated surgically, and the future of cancer treatment might not lie with the surgeon or the radiotherapist but with the biochemist.

This, however, said Dr. Robertson, was no reason for not getting super-voltage machines if there were adequate facilities and trained personnel and when a definite decision had been made which type of machine to get. It would be extravagance to buy a high-voltage machine and in a few years have to replace it with one of higher voltage. Multi-million-voltage machines were not available and a unit would have to be specially manufactured at a cost of about £75,000 for the linear accelerator, which might take as long as 2 years. It would cost more in South Africa to erect and keep going, even if trained personnel were available. Money at the present juncture would probably be far better spent in extending the availability of standard conventional radiotherapy throughout the country.

The same applied to some extent, but perhaps not quite so forcibly, to the radio-active cobalt bomb. With atomic piles cobalt bombs hundreds of times larger than the largest radium bomb in Great Britain would in due course become available. Cobalt bombs had to be renewed every 2½ to 3 years because the half-life of cobalt was only 5·3 years. The shipping and handling of these quantities of isotopes was a serious and costly matter, particularly in South Africa, 6,000 and 9,000 miles away from Great Britain and Canada, where they are produced. Radio-active cobalt gives off a ray equivalent to 2½ million volts, and its use in treatment requires highly skilled technical staff as well as experienced radiotherapists of the highest qualifications.

Another type of bomb that had been suggested was the caesium bomb; but as yet no caesium was available for South Africa, and the cost had not yet been determined.

'The outcry, then, for caesium bombs, cobalt bombs, and super-voltage radiotherapy is scarcely justified in South Africa at present when the finance involved could be used to better advantage from the point of view of doing the greatest good to the greatest number of people. If super-voltage therapy could really cure any appreciable number of patients who are otherwise incurable and not merely make the treatment better and increase the palliative effect, one would naturally strongly support obtaining these bombs and supervoltage machines as soon as possible. It must be realized, however, that the National Cancer Association of South Africa cannot of itself buy such machines.'

ITINERANT RADIOTHERAPISTS

In pursuance of a resolution taken at the 1951 National Cancer Conference, the Association made representations to the Provincial Administrations concerning the development to the highest state of efficiency of diagnostic and treatment services for cancer. Representatives of the Association were appointed to serve on a subcommittee with representatives appointed by the Johannesburg Hospital, and a comprehensive report was submitted to the Transvaal Provincial Administration on the improvement and extension of radiotherapy services in the Province.

Recently one of the subcommittee's recommendations has been implemented and 2 itinerant radiotherapists have been appointed, one for the Northern Transvaal and one for the Southern Transvaal, who visit hospitals in their areas at regular intervals to examine patients referred by their own medical attendants and to advise on treatment. Patients who can be treated locally—for instance, with radium, radon seeds or radio-active isotopes, will be treated in the outside hospitals visited. Patients requiring X-ray treatment will be referred to the Pretoria or Johannesburg hospitals for treatment.

In the Transvaal, owing to long distances and the shortage of beds in the main centres, this scheme for patients who can be treated locally has great advantages, since the patient will be treated in his own local hospital under the care of his own medical practitioner.

The centres being visited at present in the Southern Transvaal are Vereeniging, Standerton, Klerksdorp and Potchefstroom; and in the Northern Transvaal Barberton, Ermelo, Witbank and Pietersburg.

Dr. Robertson expressed the hope that the medical profession and the public would make this scheme a success. There would in future be no excuse for long delays in the diagnosis of cases of cancer.

OVERSEAS RESEARCH

He then referred briefly to world trends in the fight against cancer. Speaking of tobacco smoking and cancer of the lung he said that from statistical researches the present conclusions appeared to be that the risk of developing cancer of the lung increased with the amount of smoking. The issue was complicated by the association between the incidence of lung cancer and the density of population in cities. City air contained appreciable amounts of chemical carcinogens, and it was a reasonable hypothesis that prolonged inhalation of such polluted air, either alone or combined with smoking, might tend to be conductive to the incidence of lung cancer.

PASSING EVENTS : IN DIE VERBYGAAN

Utrecht International Fair: A medical section will be included at the Autumn Fair to be held at Utrecht, Holland, from 11 to 20 September 1956. The whole Fair will comprise 2,000 exhibits from 22 countries and will cover an area 33,000 sq. metres of net exhibition space. The medical section (1,687 sq. metres) will include medical instruments and appliances; equipment, furniture and kitchen installations for hospitals and pharmaceutical preparations.

* * *

Lede word daaraan herinner dat hulle die Sekretaris van die Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad, sowel as die Registrateur van die Suid-Afrikaanse Mediese en Tandheelkundige Raad, Posbus 205, Pretoria, moet verwittig van enige adresverandering.

Versuim hiervan beteken dat die *Tydskrif* nie afgelewer kan word nie. Dit het betrekking op lede wat oorsee gaan sowel as dié wat binne die Unie, van adres verander.

* * *

Members of the Association are asked to observe the Warning Notices published from time to time in connection with advertisements for appointments.

In the case of the Sick Fund for the Diamond Cutting Industry the holders of appointments who are members of the Association have all voluntarily resigned as the Association has withdrawn its approval of this Fund. Intending applicants are advised first to communicate with the Hon. Secretary, Southern Transvaal Branch, P.O. Box 10102, Johannesburg.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

Maybaker (S.A.) (Pty.) Limited announce the introduction of a new oral antibiotic, '*Rovamycin*' *brand Spiramycin* and supply the following information: It is a narrow-spectrum antibiotic, which was isolated from *Streptomyces ambofaciens* in France in 1951. 'Rovamycin' is markedly active against Gram-positive cocci, and only weakly so against Gram-negative bacteria. It has been found to be remarkably well tolerated by adults and children.

'Rovamycin' can be used to treat infections due to staphylococci, including strains refractory to penicillin. It is also indicated for pneumococcal and streptococcal infections, particularly those of the lung, respiratory tract and middle ear, and for the treatment of non-specific urethritis.

It is of notable interest that 'Rovamycin' does not interfere with normal intestinal flora and no case of staphylococcal enteritis or candida infection such as characterize the use of broad-spectrum antibiotics has been reported.

'Rovamycin' is of special value for the treatment of patients who cannot tolerate other antibiotics and those whose infections are due to organisms which are resistant to such substances. 'Rovamycin' is available as 250 mg. tablets in containers of 20 and 100.

BOOK REVIEWS : BOEKRESENSIES

HAEMATOLOGY

Progress in Hematology. Volume I. Edited by Leandro M. Tocantins, M.D., with 27 contributors. Pp. ix + 336. $9.75. New York and London: Grune & Stratton, Inc. 1956.

Contents: Introduction, L. M. Tocantins. 1. Gastric Intrinsic Factor and Vitamin B_{12} Interrelationships, C. C. Ungley and R. B. Thompson. 2. Parenterally Administered Iron in the Treatment of Hypochromic Anemia, Elmer B. Brown and Carl V. Moore. 3. Exchange Transfusion as a Therapeutic Measure, with Special Reference to Its Use in Erythroblastosis Fetalis, Thomas R. Boggs, Jr. 4. The Life Span of Erythrocytes and Their Post-Transfusion Survival, Max M. Strumia. 5. Abnormal Hemoglobins, Wolf W. Zuelzer, James V. Neel and Abner R. Robinson. 6. Radioactive Colloidal Gold in the Treatment of Severe Acquired Hemolytic Anemia Refractory to Splenectomy, L. M. Tocantins and George C. Wang. 7. Radioactive Phosphorus in the Treatment of Primary Polycythemia (Vera), Lowell A. Erf. 8. The autoimmune Thrombocytopenias, William J. Harrington, Virginia Minnich and Grace Arimura. 9. Guiding Principles in the Surgery of Hemophilic Patients, Anthony F. DePalma. 10. Mechanisms of Production of Acute Fibrinogen Deficiencies, Charles L. Schneider. 11. Local Environmental Factors Affecting Hemostasis in Bleeding from the Gastrointestinal Tract, Tibor Bódi, C. Wilmer Wirts and L. M. Tocantins. 12. Systemic Lupus Erythematosus and the Blood, Malcolm M. Hargreaves and Richard W. Opfell. 13. Chemotherapy of Human Leukemia, Joseph H. Burchenal and Rose Ruth Ellison. 14. The Biochemistry and Enzymatic Activities of Leukocytes in Health and Disease, William N. Valentine. 15. Hematologic Recovery from Irradiation Injury, Leon O. Jacobson. 16. Agammaglobulinemia —Congenital, Acquired and Transient Forms, David Gitlin and Charles A. Janeway. Index.

Haematology advances at a cracking pace and the average reader finds considerable difficulty in keeping up with the latest progress. He requires books such as this one to make the process relatively painless.

This book contains a number of articles written by authorities who have themselves been in the vanguard of progress; who have, by their original work, been largely responsible for the rapid increase of our knowledge of these fields. In this category one can place the very readable and authoritative chapter on the hereditary haemoglobinopathies. With new haemoglobins continually being discovered we are not surprised to hear that the letters A to I have already been exhausted (and only B has fallen by the wayside). It is almost a race to see whether we shall run out of haemoglobins before we exhaust the alphabet! But with Zielzer and Neel in charge of the chapter it all sounds delightfully simple. Similarly Harrington discourses on the auto-immune thrombocytopenias, Ungley on gastric intrinsic factor, Hargreaves on systemic lupus erythematosus and Burchenal on the chemotherapy of human leukaemia. There are 16 chapters altogether, many of which are excellent. One learns, for instance, the indications for the use of parenteral iron (and they are indeed much scantier than the frequent use of this medicament would lead one to suppose), the validity of the various methods used to measure red-cell survival, and conclusions drawn from the extensive use of radiophosphorus in polycythaemia vera. There is mention of the importance of 'heme' pigments in addition to bilirubin in the assessment of the need for exchange transfusion in erythroblastosis foetalis as well as in acute toxic states in infants and even occasionally in adults.

There are some poor chapters. A chapter on the guiding principles in the surgery of haemophilic patients, which is a very important subject and merited extensive consideration, turns out to be a discussion (and a poor one at that) on the treatment of haemorthosis in haemophilia. Unlike most of the writers in the book, who furnish extensive bibliographies and do not hesitate to give credit to other workers, the writer of this chapter, on haemophilia, gives only one reference, which he says is 'in preparation', and it refers to a subject which has been published by others on more than one occasion in the last 5 years.

There are a few other chapters which are poor, mainly because they consider unpublished, and therefore unassessed, material, in contrast with the rest of the book, where previously published material is digested and evaluated. But the blemishes are more

than compensated for by the rest of the book. It is a book that may well be read with profit, and Vol. II, if it maintains the same high standard, will receive an equally warm welcome.

C.M.

LAW OF DOCTOR AND PATIENT

Doctor and Patient and the Law. Third Edition. By Louis J. Regan, M.D., LL.B. Pp. 716. South African Price £5 6s. 3d. St. Louis: The C. V. Mosby Company. 1956.

Contents: Chapter I. Malpractice or Negligence. 1. Definition of Malpractice. 2. Malpractice Illustrated. 3. Malpractice or Negligence. 4. Chapter Discussion. Chapter II. Physician and Patient. 5. The Legal Duty of a Physician. 6. Prenatal Injuries. 7. Physician-Patient Relation. 8. The Physician is not an Insurer. 9. Care and Skill required of a Specialist. 10. Chapter Discussion. Chapter III. Special Rights to be Respected. 11. There must be Consent for Operation. 12. There Must Be Consent to Autopsy. 13. Invasion of the Right of Privacy. 14. Privileged Communications. 15. Chapter Discussion. Chapter IV. Liability for Act of Another. 16. Negligence of Partner, Employee, or Associate. 17. Chapter Discussion. Chapter V. Hospitals. 18. Charitable Hospitals. 19. Private Hospitals. 20. Public Hospitals. 21. Hospitals Not Liable for Acts of Attending Physician. 22. Right to Practice in a Hospital. 23. Chapter Discussion. Chapter VI. Expert Witnesses. 24. The Expert Witness. 25. Experts of Different Schools. 26. Hypothetical Questions. 27. Chapter Discussion. Chapter VII. Expert Testimony. 28. Expert Testimony is Usually Necessary. 29. When Expert Testimony Is Not Necessary. 30. Res Ipsa Loquitor. 31. Chapter Discussion. Chapter VIII. When Physician is Liable. 32. Proximate Cause. 33. Contributory Negligence. 34. Statute of Limitations. 35. Chapter Discussion. Chapter IX. Evidence. 36. Evidence—Admissibility. 37. Chapter Discussion. Chapter X. Proof. 38. Proof Required of Negligence. 39. No Liability for Honest Error of Judgment. 40. Admissions. 41. Nonsuit and Directed Verdict. 42. Chapter Discussion. Chapter XI. Miscellaneous. 43. Abortion. 44. Adoption. 45. Adverse Party Called as Witness. 46. Artificial Insemination. 47. Blood Grouping Tests in Affiliation Proceedings. 48. Contraceptive Statutes. 49. Contracts in Restraint of Trade. 50. Coroners and Medical Examiners. 51. Criminal Malpractice. 52. Damages. 53. Disclosure under Statute. 54. Dying Declaration. 55. Experimentation. 56. Federal Food, Drug, and Cosmetic Act. 57. Implied Promise to Pay for Professional Services. 58. Insane Persons. 59. Insurance. 60. License to Practice. 61. Medical Societies. 62. Narcotics. 63. Pharmacists. 64. Special Defenses. A. Release. B. Res Judicata. C. Compensation Award as Bar to Malpractice Action. D. Requirement of Written Notice. 65. Wrongful Death Actions. 66. X-ray Films, Ownership of. Chapter XII. The Dentist. 67. Duties and Liabilities of Dentist Analogous to Those of Physician. 68. Chapter Discussion. Chapter XIII. The Nurse. 69. General Considerations. 70. The Nurse in Industry. 71. The Nurse and Malpractice. 72. Chapter Discussion. Chapter XIV. Public Health. 73. The Law of Public Health. 74. Foeticide, Infanticide, Concealment of Birth or Death. Chapter XV. Forms. 75. Consent to Operation. 76. Consent to Operation Upon Minor. 77. Anticipatory Consent to Treat Minor. 78. Consent for Autopsy. 79. Consent to Disclosure of Privileged Information. 80. Patient Leaving Hospital Against Advice. 81. Acknowledgement of Hazards of Particular Therapy. 82. Obstetrician Unable to Be Present at Delivery. Authorization to Treat Patient Who has Recently or Partially Aborted. 84. Patient Fails to Carry Out Advice. 85. Patient Fails to Keep Appointment. 86. Patient Discharges Physician from Case. 87. Physician Withdraws From Case. 88. Patient Injured, or Alleging Injury, in Hospital. Chapter XVI. Malpractice Vulnerability, Self-Test. 89. General Questions. 90. The Physician—Personal Factors. 91. Equipment, Records, and Employees. 92. Self-Determination of Vulnerability. Chapter XVII. 93. Prevention of Malpractice Claims—Education of the Physician. 94. Prevention of Malpractice Claims—Education of the Public. 95. The Patient's Interest in Malpractice. 96. Malpractice Prophylaxis—Admissions. 97. Malpractice Prophylaxis—Records. 98. Malpractice Prophylaxis—Unethical Criticism. 99. Malpractice Prophylaxis—Tact. 100. Malpractice Prophylaxis—Liability for Act of Another. 101. Malpractice Prophylaxis—Operation, Autopsy, Hazardous Therapy. 102. Malpractice Prophylaxis—X-ray, Diathermy, etc. 103. Malpractice Prophylaxis—Insufficient Treatment. 104. Malpractice Prophylaxis—Miscellaneous. 1. Refusals. 2. Contract versus Tort. 3. Experimentation. 4. Sterilization. 5. Equipment. 6. Unwise to be too secretive. 7. Inquests. 8. Self-sufficiency. 9. Contributory Negligence. 10. Statute of Limitations. 11. Administration of Anesthetics. 12. Consultants. Chapter XVIII. Allegations in Malpractice Suits. 105. Frequent Allegations in Malpractice Suits. Chapter XIX. Case and Comment. 106. Illustrative Cases With Comments. Chapter XX. Medical Defense. 107. Strong Defense Necessary. Chapter XXI. A Malpractice Program. 108. Why a Malpractice Program? 109. Fundamentals of a Constructive Malpractice Program. 110. The Grievance Committee and Malpractice. 111. Routine Case Handling in Claims Prevention. Conclusion. Table of Cases Cited.

For the purpose of this work the author deals only with that part of the law which concerns problems which arise out of the duties, privileges and obligations of the doctor in relation to his patients. The other aspects concerning forensic medicine and its allied subjects are left to the many other books on these matters. The wide scope of the book is indicated in the contents list above. Each section consists of a discussion of the topic dealt with, so that the reader is able to gain a reasonable understanding of the matter before proceeding to the extracts of decided cases taken from court records. The selection of the extracts is designed to illustrate the points arising from the discussion and to show how decisions have varied at times.

One must bear in mind that the decisions were made in the courts of the United States and that they may not necessarily constitute precedents in the courts of this country. However they make interesting reading.

The responsibilities of the dentist and nurse are touched upon as well as the relationship between the hospital and the patient.

The writer states that 'the likelihood of being sued for malpractice is now so great that it constitutes a definite occupational hazard to the practising physician' and he points out the many difficulties that beset the doctor in his practice and the lack of mutual trust and confidence that is inspired by the possibility of legal action.

Regarding the duties of the doctor the author writes: 'Of all the obligations and responsibilities burdening the physician, the greatest is without question the duty he owes to his patient. It is in this that the real reason for his being is found. The patient expects his physician to be a dedicated person—gentle and kind, always available, inexhaustibly patient, everlastingly resourceful, and possessed of endless charity. That which is for the greatest benefit to his patient must be the first consideration of the physician'.

A.H.T.

STRESS: FIFTH ANNUAL REPORT

Fifth Annual Report on Stress—1955/56. Edited by Hans Selye and Gunnar Heuser. Pp. 815. New York: MD Publications, Inc. 1956.

Contents: Introduction. Definitions and Terminology. Part I. General Physiology and Pathology of Stress—Hans Selye. The Stress Concept in 1955. A. Brief Synopsis of the Stress Concept as It Presents Itself in 1955. B. Principal Problems of Stress Research in 1955. Introduction. I. Generalities. What is Stress? Anti-stress Drugs. II. Analysis of Pathways. (1) The Stressor and its Immediate Effects. (2) The 'First Mediator of Stress-Responses'. (3) The Hypothalamo-Hypophyseal System. A. The Hypothalamus. B. The Posterior Lobe. C. The Anterior Lobe. (4) The Adrenals. A. Afferent Nerves. B. The Adrenal Medulla. C. The Adrenal Cortex. (5) The Kidney. (6) The Nervous System. (7) The Thyroid. (8) The Ovary. (9) The Liver. III. Analysis of Chronology. IV. Analysis of Complex Disorders. A. Experimental diseases of adaptation. B. Clinical diseases of adaptation. Special Articles. Primary Aldosteronism. A new Clinical Entity. By Jerome W. Conn and Lawrence H. Louis. Hormonal Influences on Inflammation and Detoxification. By Thomas F. Dougherty and R. D. Higginbotham. Stress and Catechol Hormones. By U.S. von Euler. Adrenal influences upon the Stomach and the Gastric Response to Stress. By Seymour J. Gray, Colin G. Ramsey, Ramon Villarreal, and Lewis J. Krakauer. The Role of the Adrenal Cortex in the Etiology of Disease. By Dwight J. Ingle. Adrenocortical Secretion and Factors Affecting That Secretion. By Don H. Nelson. Neurosecretion. By Ernst Scharrer. Some Observations on Psychiatric Stress in Infancy. By Rene A. Spitz. Cortisone in Relation to Lymphoid Tissue and Immunity. By H. C. Stoerk. Part II. Special Physiology and Pathology of Stress in 1955—Gunnar Heuser. Introduction. The Order of Precedence. The Annotation of Subject. The Annotation of the Experimental Results. Reviews and Critiques. The Stressor Agents. The Adaptive Hormones. Chemistry and Pharmacology. Tests for Stress and Resistance. Resistance Diseases. Metabolic Changes: General Metabolism. Carbohydrate Metabolism. Lipid Metabolism. Nitrogen Metabolism. Salt Metabolism. Acid Base Balance. Water Metabolism. Hormones and Hormone-like Substances. Enzymes. Vitamins. Hemoglobin and Its Derivatives. Various Metabolites. Metabolic Diseases. Organ Changes: Nervous System (including psyche). Nervous Diseases. Psychic Diseases. The Endocrines and Endocrine Diseases. Blood-count. Diseases of Blood. Erythrocyte Sedimentation Rate (ESR). Blood-clotting. Thromboembolic and Hemorrhagic Diseases. Hemopoietic System. Hemopoietic Diseases. Cardiovascular System. Cardiovascular Diseases. Kidney. Renal Diseases. Respiratory System. Respiratory Diseases. Gastrointestinal System. Salivary Glands. Diseases of Salivary Glands. Liver Hepatic Diseases. Bones. Diseases of Bones. Joints. Diseases of Joints. Muscular System. Muscular Diseases. Skin and Appendages. Cutaneous Diseases. Hibernating Gland. Sense Organs and Diseases of Sense Organs. Connective Tissue. Inflammation and Wound-Healing. Connective Tissue Diseases. Microbes, Serologic Reactions. Serologic Diseases. Neoplasia. Malformations. Part III. Afterthoughts. Endotheliomyelosis—An Experimental Model of the 'Focal Syndrom'. By Hans Selye and Pierre Bois. References. Acknowledgements. Index.

The first 100 pages of this book consists of a review by Hans Selye of the present status of the stress concept. The next 100 pages contains 9 special articles, all of which bear some relationship to stress. The contributors to this section are all distinguished workers and all of them have made valuable original contributions. Because of this it is invidious to mention any particular one. One could question whether it would not have been better to publish these papers in medical journals. (One of them 'Primary Aldosteronism, a new clinical entity', by Conn and Louis, does in fact appear in the January number of *Annals of Internal Medicine*, and the topicality of the subject is sufficient excuse for reprinting it in this volume.)

The rest of the 600 pages of this book are to all intents and purposes a glorified and massive *index medicus*, which is not particularly easy to follow. Those who are unfamiliar with the terminology, abbreviations and mode of presentation require a postgraduate course to be able to follow it, and this despite extensive explanations and illustrations. The authors have not only listed and grouped the articles but have attempted to indicate the conclusions reached in the article. The latter has some slight value but it is unlikely to obviate the necessity of referring to the original publication. All in all it is a massive compilation, sur-

prisingly up-to-date, and a veritable credit to a modern filing system.

One cannot help wondering if it is all worth while. With each annual volume the number of books to be consulted increases. The field of stress as defined in this volume is now so vast that it embraces most of medicine. There is more than a germ of truth in the statement (page 220) that it would be more interesting and surprising to find a factor which fails to produce the general adaptation syndrome than to find one that produces it! One could imagine the general reader purchasing a book consisting of the first quarter of this work. A medical library or an advanced laboratory might be persuaded to purchase the rest of the book. The two halves could well be separated. More important, the necessity for producing annual volumes of this type could perhaps be considered.

C.M.

CHRISTOPHER'S SURGERY

Christopher's Textbook of Surgery. Sixth Edition. Edited by Loyal Davis, M.D. Pp. xvii + 1484, with 1,359 illustrations on 716 figures. $15.50. Philadelphia and London: W. B. Saunders Company. 1956.

Contents: 1. History of Surgery. 2. Physiology of Wound Healing. 3. Surgical Bacteriology, Chemotherapy and Antibiotic Therapy. 4. Shock. 5. Principles of Pre- and Postoperative Care: Preoperative, Operative and postoperative Care. Management of Fluid and Electrolytes in Surgical Practice. 6. Endocrinology and Metabolism in Surgical Care. 7. Anesthesiology. 8. Basic Principles of Technique in Surgical Care. 9. The Principles of the Surgical Care to the Soft Tissues. 10. Thermal and Irradiation Injuries: Burns. Local Cold Injury. Irradiation Injuries. Nuclear Radiation Injuries. 11. The Head: The Scalp. The Eyes. The Nose, Nasal Acessory Sinuses and the Pharynx. Tumours of the Nose, Nasal Sinuses and Nasopharynx. The Ears. The Mouth, Tongue, Jaws and Salivary Glands. 12. The Neck: Developmental Anomalies, Tumours, Infections and Wounds of the Neck. Larynx and Trachea. 13. The Thyroid and Parathyroid Glands. 14. The Breasts. 15. The Mediastinum. 16. The Thoracic Wall, Pleura and Lungs: The Thoracic Wall and Pleura. Diseases of the Lungs and Bronchi. Tumours of the Lungs and Bronchi. 17. The Heart and Pericardium. 18. The Abdominal Wall and Peritoneum. 19. Hernia. 20. The Alimentary Canal: Congenital Malformations. The Esophagus. The Stomach. Peptic Ulcer and Benign Gastric Lesions. The Surgical Treatment of Peptic Ulcer. Tumours of the Stomach. The Duodenum, Jejunum, Heum and Appendix. The Colon. The Anal Canal and Rectum. Intestinal Obstruction. 21. The Liver and Biliary System: Anatomy and Physiology of the Liver and Biliary System and Diseases of the Gallbladder and Bile Ducts. Diseases of the Liver. 22. The Pancreas. 23. The Adrenal Glands. 24. The Spleen. 25. The Urinary System: Methods of Diagnosis. Infections of the Urinary Tract. Anomalies and Injuries. Tumours and Calculi. 26. The Male Reproductive System. 27. The Female Reproductive System. 28. The Bones and Joints: Considerations in the Treatment of Closed and Open Fractures. Pathology and Repair of Fractures. Fractures and Dislocations. Fractures and Dislocations of the Upper Extremity and Spine. Fractures and Dislocations of the Pelvis and the Lower Extremity. Muscles, Ligaments and Bursae. Acute and Chronic Infections of the Bones and Joints. Tuberculosis of the Bones and Joints. Tumours of the Bones and Joints. 29. Surgery of the Hand. 30. Surgery of the Foot. 31. Amputations and Artificial Limbs. 32. Physical Medicine and Rehabilitation. 33. The Vascular System: The Veins. The Arteries. The Lymphatics. 34. The Nervous System: Neurosurgical diagnostic Procedures. Craniocerebral Iniuries. The Cerebrum and Cerebellum. The Spinal Cord. The Peripheral and Cranial Nerves. The Autonomic Nervous System; The Neurosurgical Relief of Pain. Congenital Anomalies. 35. The Qualifications of a Surgeon. Index.

Loyal Davis points out in his preface that this 6th edition of Christopher differs from previous editions in its presentation of the subject. Your reviewer used the 2nd edition as a student 20 years ago; while at that time the book was an outstanding one, the present edition makes much more interesting and more widely informative reading.

The editor has gathered a splendid group of contemporary outstanding American surgeons, who have endeavoured 'to place before the student an interestingly told story of the facts and principles which should form the basis for his self-education in surgery which must continue throughout his professional life'. Perusal of the list of contributors lends emphasis to the fine character of the volume, if this is required.

Whipple makes a fascinating story of the History of Surgery and Meleney's contribution on bacteriology and on chemotherapy and antibiotic therapy is no less authoritative for being exhaustive. Ravdin's chapter on shock admirably sets out the practical present-day position of this much investigated subject. Pre- and post-operative care are carefully dealt with, with nice attention to all common and several uncommon complications. Crile's section on the thyroid accurately summarizes our present knowledge of the subject—perhaps the absence of clear knowledge of malignant exophthalmos explains its omission as a topic. The section on the breast, too, is a well-reasoned exposition with a very fair distribution of emphasis on various present opinions. McVay's work on hernia is thorough and sound, as is to be expected. A glance at the authors of the section on the alimentary tract—Sweet, Baborka, Walters, Marshall and others—ensures high quality in this department.

A delightful aside on some of the duties of a houseman, of a kind not usually included in text-books, is found in 'observing the amenities' under 'basic principles of technique' in Surgical Care. 'The Qualifications of a Surgeon' carries a message for students which could profitably be read by many of us in busy practice. The book is highly recommended as an excellent advance on the now outmoded style of most modern surgical text-books.

P.C.W.M

A CLASSIC PHYSIOLOGY

Principles of Human Physiology. (Twelfth Edition.) By Sir Charles Lovatt Evans, D.Sc., F.R.C.P., F.R.S., LL.D., with chapters on the Special Senses by H. Hartridge, M.A., M.D., Sc.D., F.R.S. Pp. xii + 1233. Illustrations 721—some in colour. 65s. net. London: J. & A. Churchill Ltd. 1956.

Contents: I. Introduction. Book I. General Principles. A. Structural Principles. II. The Structural Basis of the Body. B. Biochemical Principles. III. The Elementary Constituents of Protoplasm. IV. The Proximate Constituents. C. Biophysical Principles. V. The Energy of Molecules in Solution. VI. The Properties of Colloids. VII. The Passage of Water and Solutes through Membranes. VIII. Bioelectric Potentials. IX. Enzymes and Enzyme Action. Book II. Tissues Subserving Movement and Conduction—Muscle and Nerve. Historical Note. X. Voluntary Striated Muscle. XI. Plain Muscle and other Contractile Tissues. XII. Nerve Fibres. Book III. Centralized Control and Co-ordination—The Central Nervous System. Historical Note. XIII. General Features of a Central Nervous System. XIV. The Receptors. XV. The Spinal Cord and its Relationship to the Higher Parts of the Central Nervous System. XVI. The Excitability of Neurones and the Properties of Conduction at Synapses. XVII. Characteristics of Reflex Actions. XVIII. The Labyrinth, and Postural Reflexes. XIX. Higher Afferent Paths. XX. The Cerebral Hemispheres and the Efferent Paths. XXI. Functions of the Cerebral Cortex. XXII. The Cerebellum. XXIII. The Autonomic System: The Hypothalamus: Humoral Transmission. Book IV. The Supplying of Information—The Special Senses (by F. Hartridge, F.R.S.) XXIV. The Nervous Mechanism of Sensation. XXV. Vision. XXVI. Hearing and Speech. XXVII. Cutaneous Sensations. XXVIII. Sensations of Smell and Taste. Book V. Systems for Distribution of Materials—Blood, Circulation and Respiration. Historical Note. XXIX. The Blood. XXX. General Features of the Circulation. XXXI. Physiology of the Heart. XXXII. Factors which Modify the Heart's Action. XXXIII. Dynamics of the Circulation. XXXIV. Local Regulation of Blood Flow. XXXV. The Pulmonary, Hepatic and Cerebral Circulations. XXXVI. Examples of Circulatory Adaptation. XXXVII. Lymph, Tissue Fluids, and Cerebro-spinal Fluid. XXXVIII. The Defence of the Organism against Infection. XXXIX. Respiration. (I) Mechanics of Respiratory Movements. (II) The Chemistry of Respiration. XL. The Control of the Respiratory Movements. Book VI. The Intake of Materials—Nutrition, Metabolism. Historical Note. XLI. The Exchanges of Matter and Energy in the Body. XLII. The Vitamins. XLIII. The Normal Diet of Man. XLIV. The Physiology of Digestion—Changes undergone by the Foodstuffs in the Alimentary Canal. XLV. The Absorption of the Foodstuffs. XLVI. The History of the Foodstuffs. Book VII. The Removal of Waste Material and Temperature Conservation—Excretion and Temperature Regulation. Historical Note. XLVII. Renal Excretion. XLVIII. The Skin and the Skin Glands. XLIX. The Temperature and Heat-Balance of the Body. Book VIII. The Provision of Special Chemical Stimulants, and of Measures for Continuance of the Species—The Endocrine Organs and Reproduction. Historical Note. A. Endocrine Organs. L. The Endocrine Organs. B. Reproduction. LI. The Formation of Germ Cells. LII. Reproduction in Man and other Mammals. LIII. The Secretion and Properties of Milk. LIV. Heredity.

Originally by Starling, written since 1930 by Lovatt Evans, this remains the standard British text-book of physiology. It is a great achievement that it should still be written almost entirely by one man. This single authorship gives the book a unity of thought which is lacking in many composite text-books and there is little to support the author's statement, expressed in the preface to a previous edition, that it makes the book 'patchy, superficial and out of date'.

Inevitably the single authorship of the greater part of the work precludes the detailed information on some of the most recent advances in each field which could be provided by a team of specialists, but this is not a major fault, since it limits the book to the general information which is required by every physiologist. This general information is commendably up to date.

In general, space is reasonably allocated to the several systems but the chapters on blood and on the endocrine organs are rather condensed and the description of blood coagulation omits several important factors. The chapters on sensation, as in previous editions, are written by Professor Hartridge, whose contribution fully maintains the high standard of the rest of his work. The biochemical principles enunciated are adequate for the reader's

understanding of the physiology described, but this is not a text-book of biochemistry and the details of that subject must be sought elsewhere.

Lovatt Evans is not a book to be recommended to students commencing the study of physiology. It is so full of information that the novice may have difficulty in appreciating the general pattern. For the more advanced student and as a reference book on physiology for biologist or medical practitioner it has never been surpassed.

A.W.S.

CORRESPONDENCE : BRIEWERUBRIEK

THE DISPENSING DOCTOR

To the Editor: In accordance with the planned propaganda scheme initiated by Mr. Kramer on 9 April 1956, the Pharmaceutical Society and the Pharmacy Board continue their attacks on the medical profession in the public press. The public are being told (*a*) that protected by law, the doctors are in competition with the chemists and (*b*) that in consequence there is a shortage of chemists. This is illogical, for if there is a shortage of candidates for Pharmacy, how is the public to benefit by inhibiting the dispensing doctor? There is no evidence that doing so will entice students to pharmacy or that the dispensing doctor does in fact make young men and women hesitate to take up pharmacy. It is not the doctor (or the pharmacist for that matter) who is protected, but the public—from the handling of drugs by untrained people. And it is the public's interest *and theirs only* that has to be considered.

Doctoring is a very personal application of the science and art of healing, and it has to be carried out on the cultural level of the patient. To country people and the lower classes in town this doctoring includes dispensing by the doctor. It is not merely the doctor's rights the pharmacists seek to limit, but the patient's right of free choice of dispensing doctor. If these patients cannot be attended by the dispensing doctor they will inevitably by-pass the doctor and drift to the counter-prescribing chemist.

It is a pity that the clause seeking to limit doctors' legitimate dispensing services to the public should have been withdrawn, for this served only to emphasize the pharmacists' deplorable propaganda. Parliament consists of the representatives of the people and—in defence of the rights of the people, the patients, the sick—I have no doubt that Parliament would have thrown out the clause and so put an end to the matter and to the unpleasant tactics of a medical auxilliary group who should be in cooperation with not antagonistic to the medical profession. It is time that pharmacists realized that their attempts to take the place of the doctor by displacing the dispensing doctor will never receive the sanction of those responsible for the public health.

G.P.

13 August 1956

'MEDICAL REPRESENTATIVES'

To the Editor: I should like to raise a controversial subject of interest to the majority of us—that of the so-called 'medical representatives'. The term is a misnomer, because they do not represent our interests in the least. Their numbers have of recent years increased and I submit that they are wasting a great deal of our time.

Considering the continuous bombardment with printed matter and blotters we are receiving by every mail, this constant stream of men and women is unnecessary, however charming some of them are. (I have had 3 of them calling on me in 2 days!)

Those of us who read our journals and periodicals are in no need of 'sales talk'. Those whose postgraduate study has been restricted to factory 'literature' and these 'sales talks', may be satisfied to 'learn' about the 'latest on the market', even if there is as yet no report from our *alma mater* or any equally respected source on the latest 'wonder preparation'. At the Annual Meeting of the B.M.A. at Brighton Dr. Desmond Curran (London) is reported to have used the phrase '. . . the physician might be confused by the plethora of drugs now being released on the market, often without adequate clinical trials' (Brit. Med. J., 21 July 1956, page 157). I should like to add that the plethora is of drugs that are not always harmless.

I am of the considered opinion that this constant experimentation on our patients with unknown, nebulous and ephemeral preparations is unethical and highly undesirable. It is detrimental to the individual patient and uneconomical both to him and to the country as a whole. I submit that the ethical principles of this practice should be considered by our Medical Council. The Federal Council might perhaps find a *modus vivendi* to protect us from those innumerable callers; otherwise many of us will be obliged to instruct our receptionists to exclude representatives of drug houses from our consulting rooms—before they become 'insulting rooms' in spite of the many samples of 'tranquillizers' left with us! The disposal of the accumulation of 'samples' in my rooms has become a major problem.

Physician

17 August 1956

RMO'S REMUNERATION

To the Editor: I am a Railway Medical Officer in a small hospital town, about 120 miles from the nearest centre. For the past year I have kept records of the work devolving on me as RMO and feel that the figures resulting will be of interest to many RMOs in a similar position to myself.

My remuneration last year was: Salary £244, travelling allowance £12, surgical allowance £35. The number of members was 87, and of dependants 237.

I kept records, which are about 95% accurate, of the work undertaken over the year. The results are: Europeans—visits 313, consultations 1,025, total 1,338; Natives—consultations 168, total 1,506.

At my salary this works out at:

Europeans: £244 ÷ 1,338 = 3s. 7¾ each, per consultation or visit.

Natives: £30 ÷ 168 = 3s. 7d. each, per consultation or visit.

Total: £274 ÷ 1,506 = 3s. 7½d. each, per consultation or visit.

These figures do not include visits to patients in hospital, medical or surgical. In addition, my surgical allowance of £35 covered 16 minor operations (tonsils and adenoids, fractures, and dilatation and curettage, and 11 major operations (abdominal, appendix, hernia and Caesarian section). Total £35 ÷ 27 = £1 5s. 0d. odd per operation.

I have not included in the above figures major X-rays undertaken (intravenous pyelograms and cholecystogram), numbering 6. I have kept no records of minor X-rays (chest and limbs).

Confinements numbered 10 at £5 5s. 0d. = £52 10s. 0d.

This small town may be cited as a fair example of the many small towns far enough from the larger centres to make it necessary for the RMO to do many of his own investigations and major treatments. We do not get adequate remuneration for the basic work of consultation and visits; but the remuneration for hospital work (medical and surgical) is hopelessly inadequate. To ask a man to do major surgery at 25s. a time is, I feel, exploiting the working class.

My figures bring out another interesting fact: excluding hospital treatments, my 237 dependants have seen the doctor on 1,336 occasions during the year—an average of almost 6 times per dependant. Surely a record of which any community may feel proud!

To close, I do not feel that my town has more work than any other centre except that, in common with other small towns, we do major undertakings and investigations that would be cared for by the specialist in the larger towns.

I hope that the figures I present will stimulate a little argument, and that in the course of time pressure will be brought to bear by the various RMO Associations to deal with what I feel is a justifiable grievance. It has been suggested to me that people in a similar position to myself be awarded a disability (distress) allowance to cover the extra work involved.

J. H. Youngleson

P.O. Box 112
Kokstad
3 August 1956